DISSERTATION ON

EFFICACY OF ALPHA LIPOIC ACID IN ADJUNCT WITH INTRALESIONAL HYDROCORTISONE AND HYALURONIDASE IN THE MANAGEMENT OF ORAL SUBMUCOUS FIBROSIS

M.S. DEGREE EXAMINATION BRANCH - IV

OTORHINOLARYNGOLOGY

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CERTIFICATE

This is to certify that the dissertation entitled **EFFICACY OF ALPHA LIPOIC ACID IN ADJUNCT WITH INTRALESIONAL HYDROCORTISONE AND HYALURONIDASE IN THE MANAGEMENT OF ORAL SUBMUCOUS FIBROSIS** is a bonafide record of work done by **Dr. AMIRTHAGANI. A** in the department of Otorhinolaryngology, Thanjavur Medical College, Thanjavur, during his post graduate course from 2010-2013 under the guidance and supervision in partial fulfilment for the award of M.S. DEGREE EXAMINATION BRANCH - IV (OTORHINOLARYNGOLOGY) to be held in April 2013 under **The Tamil Nadu Dr.M.G.R.Medical University, Chennai.**

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I declare that this dissertation entitled **"EFFICACY OF ALPHA LIPOIC ACID IN ADJUNCT WITH INTRALESIONAL HYDROCORTISONE AND HYALURONIDASE IN THE MANAGEMENT OF ORAL SUBMUCOUS FIBROSIS"** is a record of work done by me in the department of Otorhinolaryngology, Thanjavur Medical College, Thanjavur, during my Post Graduate Course from 2010-2013 under the guidance and supervision of my unit chief **Prof. Dr.A.RAVINDRAN, M.S., D.L.O, Associate Professor,** and **Professor and Head of the Department Prof. Dr.T. RAMANATHAN** MS., D.L.O. It is submitted in partial fulfilment for the award of **M.S. DEGREE EXAMINATION – BRANCH IV (OTORHINOLARYNGOLOGY)** to be held in April 2013 under **The Tamil Nadu Dr.M.G.R.Medical University, Chennai.** This record of work has not been submitted previously by me for the award of any degree or diploma from any other university.

Place: Thanjavur
Date :

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CONTENTS

ABBREVIATION

OSMF	:	Oral Submucous Fibrosis
ALA	:	Alpha Lipoic Acid
NON-ALA	:	Non Alpha Lipoic Acid
DCP	:	Differential Count Polymorpho Nuclear Leukocytes
DCL	:	Lymphocytes
DCE	:	Eosinophils
DCM	:	Monocytes
HB	:	Hemoglobin
BL	:	Betal Leaf
BN	:	Betal Nut

1. INTRODUCTION

Oral sub mucous fibrosis is a precancerous lesion, predominant condition seen in South Asian ethnic group ,more in India .No age or sex predilection has been found. OSMF is primarily a cell mediated immune reaction to areca nut chewing and betel quid. It is prevalent in people belonging to poor socioeconomic status associated with nutritional deficiency.

In 1953 S.G.Joshi termed this condition as Oral sub mucous fibrosis. And latter it was described by Pindborg and Sirsat in (1966).[1]

In simple words Oral sub mucous fibrosis is a chronic progressive crippling fibrotic but preventable disease.

Manifestations are dryness and burning sensation in the mouth, blanching of oral mucosa and reduced mobility of tongue.

It is a chronic pre-cancerous condition leading to juxtaepithelial fibrosis mainly at Retro molar trigone and Soft palate.

Young people are attracted by the media publicity of areca nut preparations thereby getting addicted to the same. There are 5 million young Indians affected from this crippling disease.

Most important aspect of the treatment is cessation of the habits. Intralesional steroids are commonly used, but the dose and duration of the treatment have not been standardized so far. Other treatments include

placentrex, hyaluronidase, collagenase, elastase and oral zinc and pentoxiphylline.

Five of the study population including two school students are below the age of 20, since there is no restriction for sale of areca products in public places, these age groups are more vulnerable for addiction.

There must be careful observation and long term follow up,

Patients are instructed that at any time it may recur and may worsen even to malignancy.

2. JUSTIFICATION

Young patients are chewing processed areca nut due to a change in lifestyle leading to adoption of the habit at a younger age.

In this study five are less than 20 years of age, among them two are school going age. Less cost, easy availability and sale in prohibited areas like public places, near schools and colleges makes young people vulnerable for addiction.

OSMF causes restriction in mouth opening, reduction in food intake, patient become malnourished and anaemic.

School students and College students are also getting addicted to these chewing habits even after knowing the seriousness of the addictive quality and the danger of oral cancers.

In this study all the patients were strictly advised to stop the oral abusive habit at the beginning itself. And there must be a careful observation and follow up for each and every case for at least one to two years.

All the available treatment for OSMF is only a minimal improvement and symptomatic relief that is why this study was conducted to evaluate the efficacy of oral Antioxidant therapy by using Alpha lipoic acid.

In a study to evaluate the effectiveness of Alpha lipoic acid (ALA) by randomized double-blind placebo-controlled trial there were significant improvements in the symptoms notably burning sensation of mouth.[2]

Areca nut and tobacco are the precipitating factors. Preventive measures and habit restriction should be followed in all clinically suspected cases to retard the progression of disease. In South-East Asian countries the habits of consuming tobacco, betel nut are more in India. The reason for this is majority of Indian population depend on agriculture for their living and income. After working in fields, they take these substances as light food, till they reach home.

It is easily available in their work places. When people are asked, they say there is a feeling of satiety in between meals when they have this. Some others have a sense of well-being. One other pathetic condition is that they take it some five to ten times in a day. In villages young people and children in their home also start taking this automatically since they are constantly exposed to their parents and grandparents who are habitual chewers.

When the above scenario is going on one side, the people in town and cities are exposed in a different way. They have a different attitude towards chewing. Some people take it for bad breath.

In still more developed area, the estimation is like that of a fashion among youngsters to chew this. They get these substances readily from shops

nearby. Even though there is prohibition to sell nearby Educational institutions, it is not being followed strictly.

More over Thiruvaiyaru taluk in Thanjavur district is well known for betel plant, seeval (betel nut product). And tobacco factories are there in Thanjavur. People here have the habit of chewing when they get together for functions.

3. AIMS AND OBJECTIVES

1. To study the clinical features of the patients with Oral submucous fibrosis attending the outpatient department of Otorhinolaryngology Thanjavur Medical College Hospital, Thanjavur.

2. To measure the Efficacy of Alpha lipoic acid in the management of Oral submucous fibrosis.

4. REVIEW OF LITERATURE

I. ANATOMY

II. AETIOLOGY

III. EPIDEMIOLOGY

IV. PATHOGENESIS

V.PATHOLOGY

VI. CLASSIFICATION AND STAGING

VII. COMPLICATION

VIII. TREATMENT PRINCIPLES AT VARIOUS LEVELS

IX.ARECA NUT AND ITS ADVERSE EFFECT

X. ALPHA LIPOIC ACID (ALA)

XI.OTHER PREMALIGNANT AND MALIGNANT LESIONS OF ORAL CAVITY

I.SURGICAL ANATOMY OF ORAL CAVITY AND OROPHARYNX.

ORAL CAVITY[3]

Outer part is called vestibule and the inner part is called oral cavity proper.

LIPS

Upper and lower lips are lined by skin in outer part, mucous membrane inside. It consists of skin, superficial fascia, orbicularis oris and submucosa,

CHEEKS

Present on either side of oral cavity

It Consists of skin, fascia,buccinators ,submucosa and mucous membrane.

ORAL CAVITY PROPER

BOUNDARIES

Roof of the oral cavity is formed by palate,

Floor is formed by Tongue

Posteriorly it is opening into pharynx

Anteriorly it is bound by teeth and gums

GUMS**:** Tissue surrounding the alveolar process of the jaw, it contains fibrous tissue.

It has two parts

1. Loosely encircling neck of tooth a collar,
2. Firmly attached to alveolus.

HARD PALATE: Separates nose and oral cavity, formed by palatine process of maxilla anteriorly and palatine bone posteriorly

SOFT PALATE**:** it is a muscular structure hanging from posterior part of hard palate, separates nasopharynx and oropharynx.

ANATOMY OF RETROMOLAR TRIGONE

It is a triangular area of mucosa overlying ascending ramus of the mandible .Apex is formed by maxillary tuberosity and the base is formed by last molar tooth.

Laterally it continues as gingivobuccal sulcus and medially as gingivolingual sulcus.

Since mucosa adhere closely to the mandible, if any malignancy occurs in this area will infiltrate easily in to the mandible and inferior alveolar nerve.

It is divided in to the following components,

OROPHARYNX

ANTERIOR WALL

It is formed by posterior third of tongue posterior to foramen caecum and vallecula.

LATERAL WALL

It continuous as palatoglossus muscle, palatopharyngeus muscle and palatine tonsil.

ROOF

Formed by soft palat**e.**

POSTERIOR WALL

Anterior to second and third cervical vertebra.

The lymphatic drainage of Oropharynx is mainly in to

1. Jugulodigastric nodes,
2. Retropharyngeal nodes and
3. Parapharyngeal nodes.

NORMAL HISTOLOGY OF ORAL MUCOSA[4]

Mucous membrane of the oral cavity consists of stratified squamous epithelium and the connective tissue supporting mucosa is called lamina propria.

In the oral cavity, there is no clear distinction between lamina propria and sub mucosa.

The oral mucosa varies from site to site within the oral cavity, but the epithelium is stratified squamous at all the sites. This epithelium is partially keratinized on gingiva and hard palate and on tongue, non-keratinized in other areas of oral cavity. Oral epithelial tissue continuously undergoes reproduction and new cells replace the dead cells.[5]

II. EPIDEMIOLOGY

Incidence: *sudhakar vaidya has stated in world article in ear, Nose and Throat as*[6]

Incidence - *4/1,000 adults in rural India*

5 million young Indians are affected due to increased use of gutkha and pan masala[7]

The disease is predominantly seen in Asian countries, prevalence being more in India.

OSMF is also reported in South Asian origin living outside, but very rare in White population.

III.AETIOLOGY

1) **Areca nut** is otherwise called areca catechu; the seed is called areca nut. It grows commonly in tropical part of pacific, Asia and parts of east Africa.[8]

2) Tobacco chewing: it is chewed alone or it is combined with betel nut, betel leaf and lime.

3) **Pan Masala:** Areca nut is the main constituent of all 'pan masala' that may or may not contain tobacco.[9]

4) Gutkha: It is prepared from betel nut and tobacco, it is sweet in taste. gutkha and pan masala are on greatest use in recent days especially among youth .

5) Taking Spicy food

6) Nutritional deficiencies: in the form of anaemia, chronic iron deficiency and vitamin B deficiency synergize progression of the disease.

According to the case report, British Dental Journal 191, 130 (2001) .Nutritional deficiencies, Iron, B-12 considered as predisposing factor for OSMF.

7) Low Socio Economical Status: OSMF is prevalent in people of Low Socio Economical Status.

8) **Immunological disorders***:* Raised ESR and globulin levels are indicative of immunological disorders. Serum immunoglobulin levels of IgA, IgG and IgM are raised significantly in OSMF. These raised level*s* suggest an antigenic stimulus in the absence of any infection. Circulating autoantibodies are also present in some cases of OSMF. [10]

9) Genetic disposition:

HLA 10, DR 3and DR 7 has been noted in OSMF [11]

10) Poor oral hygiene

11) Streptococcal infection

12) Localised collagen disease

13) OSMF is also seen among few non -Areca nut chewers.

Causes

1. Immunological process and
2. Genetic components

In those patients increased level of pro-inflammatory cytokines are found.

IV. PATHOGENESIS:

OSMF results from increased production of collagen by fibroblasts. In addition to this there is decreased breakdown leading to accumulation of excessive amount of collagen.

I. INCREASED SYNTHESIS OF COLLAGEN[12]

Arecoline and Arecadine are present in the areca nut stimulate fibroblast. Some Other growth factors also stimulate fibroblast. The only cytokine with anti-collagen effect is interferon, which is decreased in OSMF

II. DECREASED BREAKDOWN

Chewing areca nut increases copper level that stimulates fibrogenesis Tannin in betel nut stabilise collagen by cross linking effect.Furthur type III collagen is converted to type I collagen which is relatively resistant to degradation.

Both tannin and copper makes collagen highly resistant remodelling and phagocytosis.

Areca Nut

1. Alkaloids ⟶ Fibrosis ⟶ increase collagen synthesis.

2. Tannins & catechins ⟶ stabilize cross link between collagen & elastin molecules.

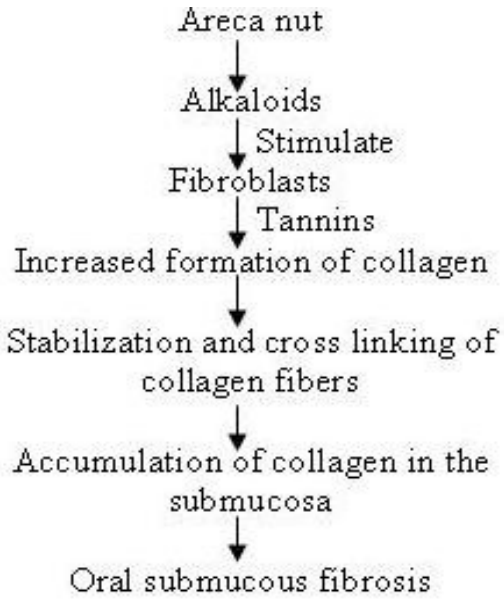
3. Copper increases collagen formation, decreases the phagocytosis.

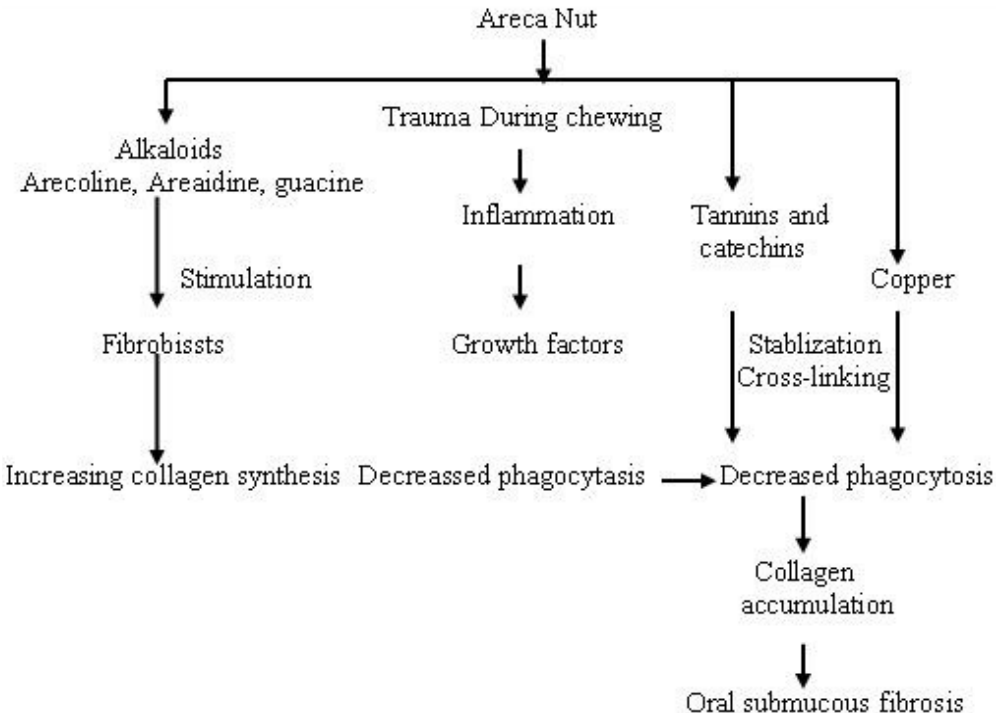
4. Trauma during chewing ⟶ Inflammation ⟶ growth factors.

Tobacco gives polycyclic aromatic hydrocarbon. It is changed into a carcinogen with the help of Aryl hydro carbon hydroxylase.

Flavanoids and tannins resist degradation caused by collagenase.

Arecolin ⟶ Fibroblast proliferation ⟶ Collagen synthesis[13]





CLINICAL FINDINGS

1. Constant dryness of mouth

2. Burning sensation of mouth

3. Intolerance to chillies and spicy food

4. Repeated vesicular eruption,

5. Ulceration,

6. Difficulty in opening the mouth and

7. Difficulty in protruding tongue.

Changes of OSMF are most marked over soft palate, faucial pillars and buccal mucosa.

In the initial stage there will be redness and formation of vesicles, which will rupture and form superficial ulceration,

In the later stage there will be blanching of mucosa after that fibrosis will develop, fibrotic bands will be seen in the affected areas. It looks like ‘marble’

Fibrosis and scaring extends in to the underlying muscles leading to restriction of mouth opening and further restriction by involving soft palate and tongue.

Trismus is gradually progressive and patient may not be able to open his mouth and put his finger or brush to clean his teeth and also there will be restriction to the tongue movement that prevents self cleaning action of the tongue over the teeth, thereby oral hygiene is badly affected and teeth become caries.

As the disease progresses, the examination of oral cavity will also become difficult to rule out other pre-malignant lesion or malignancy.

Expression of heat shock protein -70 in OSMF and oral cancers. It is a useful biomarker. This heat shock protein is synthesised during stress situation.

Thus stress plays important role in predispose OSMF, oral squamous cell carcinoma, most of the studies reveal there is increase in the incidence of OSMF.

Increase in prevalence among young generation threatening oral cancers in the future. In early stage it involves only the buccal mucosa at retromolar trigone.

In advanced cases it involves soft palate, uvula may get atrophied, tongue and lips.

There will be restriction of tongue movement and self cleaning action of the tongue is affected, oral hygiene is also affected streptococcal infection causing more and more damage to the oral mucosa.

OSMF considered as an analogue of Plummer- Vinson syndrome.

Oral candidiasis also will promote the malignant transformation by nitrosamines.

Rate of malignant transformation of OSMF is 7-13% as per the previous studies. Incidence of squamous cell carcinoma is higher in OSMF.

High frequency of epithelial dysplasia and higher prevalence of Leukoplakia in OSMF noted. According to the five criteria's given by pindborg for diagnosing precancerous condition.

Ulceration and burning sensation of mouth leads to less consumption of food leading to anaemia, it is a vicious cycle.

OSMF is an inflammatory disorder. Biopsy taken from the buccal mucosa is showing cyclo-oxygenase expression. It is mediated by prostaglandins. And inflammatory cells are seen in initial stage and later it will disappear.

Even though OSMF is being diagnosed clinically, Biopsy is mandatory to confirm the diagnosis and to rule out dysplasia and malignancy.

Muscle stretching exercises should not be more violent, this must be gentle, so that it may not give adverse effect on already affected muscles.

Heavy force used to crush the hard nut in betel nut chewers will produce muscle damage.

VI. CLASSIFICATION AND STAGING

Oral submucous fibrosis is clinically divided into 3 stages (Pindborg J.J.)[14]

Stage 1: *Stomatitis*

Stage 2: *Fibrosis*

a- Early lesions, blanching of the oral mucosa

b- Older lesions, vertical and circular palpable fibrous bands in and around the mouth or lips, resulting in a mottled, marble-like appearance of the buccal mucosa

Stage 3: *Sequelae of oral submucous fibrosis*

A. Leukoplakia

B. Speech and hearing defects

HISTOPATHOLOGICAL STAGING *OF OSMF*[15]

VERY EARLY STAGE (GRADE I)

Finely fibrillar collagen, dispersed with marked oedema, fibroblastic response is strong, blood vessels are sometimes normal, but more often they are dilated and congested, inflammatory cells, mainly polymorphonuclear leukocytes with an occasional eosinophil are present.

EARLY STAGE (GRADE II)

Juxta epithelial area shows early hyalinization, plump young fibroblasts are present in moderate numbers. Blood vessels are dilated and congested, inflammatory cells are mostly mononuclear lymphocytes, eosinophils and occasional plasma cell

MODERATELY ADVANCED STAGE (GRADE III)

Collagen is moderately hyalinised. Fibroblastic response is less marked, cells present being mostly adult fibrocytes, blood vessels are normal or constricted, inflammatory exudates consist of lymphocytes and plasma cells, although an occasional eosinophil is seen.

ADVANCED STAGE (GRADE IV)

Collagen is completely hyalinised.Hyalinized areas are devoid of fibroblasts. Blood vessels are completely obliterated or narrowed. The inflammatory cells are lymphocytes and plasma cells.

VII.COMPLICATION

1. Trismus leads to poor food intake causing malnourishment and anaemia which will further worsen the subepithelial fibrosis and trismus.
2. Nutritional deficiency will synergize OSMF.[16]
3. Unable to move the tongue, so failure to clean the oral cavity after taking food.
4. Ankyloglossia.
5. Speech and Hearing problem.
6. 3 to 10% of people having OSMF are at risk of malignant transformation.

VIII. TREATMENT PRINCIPLES AT VARIOUS LEVELS

A. NON PHARMACOLOGICAL TREATMENT

1. To stop chewing areca nut and tobacco

2. Avoid taking spicy foods, including chillies, fast food

3. Supplementing diet rich in vitamins and minerals

4. Maintaining Proper oral hygiene

5. Forgoing alcohol

6. Avoid taking hot fluids like tea, coffee

7. Encouraging jaw opening exercises by using dental screw or wooden pamparam (in Tamil)

B. MEDICAL LINE OF MANAGEMENT

1. Placental extract,

2. Trypsin,

3. Collagenase,

4. Hyaluronidase

5. Elastase

6. Intralesional Interferon-γ.[17]

7. Triamcinolone

8. Oral zinc

9. Oral pentoxiphylline.

10. Fibrinolysin and

11. Gold

12. Submucosal injections of hydrocortisone 100 mg once in a week for 12 weeks.[18]

13. Vitamins A, B-complex, c and iron.

Iron is needed for angiogenesis, Cytochrome oxidase in iron required for normal epithelial maturation.In iron deficiency anamia ,there will be atrophy of epithelium and decrease in vascularity ,which will lead on to dryness of mouth.

14. *Oral nutritional supplement of Lycophene 16 mg daily .the effect can be enhanced by intraletional Betamethasone*"[19]

15. Stem Cell Therapy [20]

16. Chymotrypsin

Hyaluronidase and steroid used in combination gives better long term result than either of them used alone.

C.SURGICAL TREATMENT

Aim of the surgical procedure: To get adequate mouth opening.

Anaesthesia **:** GA (General Anaesthesia) Naso-tracheal intubation

Maxillary, mandibular occlusal splint used mouth gag applied to obtain splint to split distance of 35mm

1. Surgical excision of fibrotic band with submucosal placement of fresh human placental graft[21]
2. bilateral tongue flaps
3. Nasolabial flap
4. Island palatal mucoperiosteal flaps
5. bilateral radial forearm free flap
6. Surgical excision and buccal fat pad graft[22]
7. Superficial temporal fascia flap and split skin graft
8. Coronoidectomy and temporal muscle myotomy
9. KTP-532 laser release procedure [23]
10. Nowadays Co_2 laser is preferred

Advantages

It cuts precisely and

Stop bleeding

Wound healing without contracture in laser excision

To avoid recurrence of trismus after surgery, inter positional graft are used. Removal of Fibrotic band by surgery may result in further scar formation.

Post Operative care

- Ryle's tube feeding
- Oral hygiene
- Mouth opening exercise – Heister's mouth gag
- Normal saline mouth wash / irrigation.

D.PHYSIOTHERAPY

1. Heat therapy- Hot rinse by Luke worm water

2. Microwave therapy -Selective heating at subepithelial connective tissue [24]

E. Nutritional support

1. High protein diet, Iron, Vitamin A, B complex, C, D and E, iron, Copper, calcium, Zinc, Magnesium, Selenium, Protein diet and milk.[25]

IX.ARECA NUT AND ITS ADVERSE EFFECT

Areca nut: it is unhusked whole fruit of the areca nut tree.

Betel nut: it is inner kernel (seed), after removing husk.

Commercially prepared pan masala, Gutkha have been marketed widely for the past 20 years. [26]

Pan Masala, Gutkha Industries have risen very shortly and quickly.

Areca in any form is risk for health.

There are two varieties in **betel nut** preparations.

They are

1. Plain variety
2. Sweet variety

Plain variety (astringent)

Cardamom and tobacco are added.

Sweet variety

It consists of Cardamom, cloves, coconut, sugar crystals, camphor, amber, nutmeg and colouring agents.

Supari

Roasted and flavoured pieces of areca nut

People of north east part of India using '**TAMOL**' the fermented areca nut. In our country tobacco is added in the quid.[27]

Since areca nut is chewed wrapped with betel leaf, it is called betel nut. It has central nervous system stimulation and taken at the end of meal for digestion and breath of mint.

Since the areca products are inadequately labelled, bearing no health warnings in the labels and no restriction on sale to students and children makes people vulnerable to these chewing habits.

Sliced and chewed betel nut will directly irritates the mucosa and sharp edges of the betel nut will cause more damage and force used to crush the hard nuts.

Betel quid chewing is more common in women than males.

It has got social acceptance in our society.

It consists of betel nut, betel leaf and slaked lime.

Betel nut is one of the commonly used psychoactive substances among rural as well as urban population our country.

Copper in areca nut

1% Bordeaux sprays is used to prevent fungal infection to areca plant. This solution consists of copper sulphate. Incorporation of copper into the areca nut is by spraying the Bordeaux mixture on areca plantations.

Copper level increase in salvia 15-30 nuts of chewing areca nut, local absorption of copper by transmucosal transport through epithelial cells, as non-enzyme dependent diffusion (Metallothionein protein), leading onto collagen formation and increase in lysyl oxidase activity, it further leads to cross linking of collagen and elastin molecules, thereby reduces the action of collagenase and prevent breaking of collagen.

Copper content

CONSTITUENTS	COPPER
Red areca	18.3 ppm
White areca	14.9 ppm
Betel leaf	18.5 ppm
Gutkha	13.2 ppm
Flavoured areca	12.2 ppm
Tobacco	6.3 ppm

Betel leaf consists of highest amount of copper

Alkaloids in betel nuts are irritating the mucosal surface, and damage the epithelium and causing juxtaepithelial fibrosis and thereby leading onto trismus.

The compositions of various areca nut preparations are different from one another according to the place where they are prepared.

Alkaloid content can be reduced in the betel nut by soaking and boiling processes.

Areca nut was used in Gujarat state in India mostly in the form of ***mawa***, a mixture of tobacco, lime and areca nut. And 10.9% of *mawa* users had OSMF. The study conducted in Gujarat state in India says that "*increased* use of areca nut products in younger age groups leads OSMF and increase the incidence of oral cancer in the future."

M.K. Gupta*, Shubhangi Mhaske, Raju Ragavendra have recently suggested that *"there is dual action of areca nut. It issuggested that arecoline not only stimulates fibroblastic proliferation and collagen synthesis but also decreases it's breakdown. This suggests that arecoline is the active metabolite in fibroblast stimulation*". [28]

Areca nut preparation and Betel quid chewing are practised predominantly in South Asia and in India, the risk of oral cancer increased with the duration and frequency of the habit.[29]

ADVERSE EFFECTS FROM CHEWING ARECA PRODUCTS

A) The areca nut contains

1. Tannins
2. Arecatannins
3. Gallic acid
4. Arecolins
5. Arecaidines and
6. Guvacine

Last three are alkaloids and all are having vasoconstriction properties.

D) Betel nut chewing will produce exacerbation of bronchial asthma and elevation of blood pressure[30]

E) There may be **chances of malignancy** in internal organs [31]

F) There is a risk of getting type **2 diabetes mellitus**.[31]

G) Areca nut chewing in psychiatric patients further worsens the condition[32]

H) During pregnancy significant increase in the adverse outcomes for the baby. Incidences of **lower birth weight**, **pre term** were found to be high. There is an 11 fold increased risk of malignant transformation in persons who chew betel quid. This chewing habit has become an addiction.[33]

As Gupta prakash explains "*While chewing they used to take a few slices of the areca nut wrapped in a fresh betel leaf along with lime (not the citrus fruit named lime), some people may add clove, cardamom and catechu".*[34]

Arecolins will interfere with the ECM – Extra Cellular matrix molecules commonly collagen. Phagocytic activity caused by fibroblast is prevented.

There will be increased secretion of inflammatory cytokines, Growth factors and decreased production of anti-fibrotic cytokines.

Important feature of OSMF is Juxta epithelial fibrosis affects most of the parts of the oral cavity, pharynx, upper 1/3 of oesophagus including pyriform sinus. Thereby leading to dysphagia and progressive trisums.

Among all the alkaloids (Arecoline, arecaidine, Guvacine and Guavacoline) Arecoline is the important agent.

It has got parasympathetic activity which gives Euphoria and counteracts fatigue.

Chronic exposure to those irritants will lead on to malignant transformation. Chewers are taking copper 5mg/day in areca nut.

Tannic acid, Arecolins in the betel nut and Calcium hydroxide ($Ca(OH)_2$) added as slaked lime are affecting the vascularity or the buccal mucosa. Tannin will synergize this action. [35]

In areca nut chewers' Excessive activity of muscles due to repeated chewing and using heavy force to crush the hard nut.[36]

There is already reduced blood supply. Increased muscle activity and reduced blood supply causing muscle degeneration and fibrosis.

The fibrosis and less vascularity with altered cytokine activity make the environment for carcinogenic tobacco and area nut to act upon the epithelial surface. These carcinogens will act for a longer duration separately.

Atrophic epithelium leads to intercellular oedema then epithelial atypia and letter it will turn into epithelial hyperplasia.In this stage malignant transformation may take place at any time.

OSMF will not regress on its own or after stopping chewing habits.

Once the disease sets in, it persists or progress.

And it involves adjacent areas like soft palate, tongue and even down up to pyriform fossa.

X.ALPHA LIPOIC ACID (ALA)

Alpha Lipoic Acid (ALA) is an anti oxidant found in fruits and vegetables like Green Pepper, Tomatoes, Grapefruit and Lemon/Limes[25]

ALA fights with free radicals .free radical damages cells tissues and organs in our bodies ALA is both water and fat soluble

Green pepper is high in alpha lipoic acids, according to the George Mateljan Foundation. In addition, green peppers are good sources of vitamin C, thiamine, vitamin B6, beta carotene and folic acid.[25]

Alpha lipoic acids have been found in tomatoes and are commonly synthesized for use in skin creams. Tomatoes also contain the powerful antioxidant lycopene. Lycopene protects from certain cancers as well as protecting the heart.[26]

Lemons and limes contain alpha lipoic acids and can be cooked or ingested raw. Lemon and limes contain limonoids, a compound that has been shown to help fight cancers.

The alpha lipoic acids found in grapefruit Grapefruit's high antioxidant activity can be ingested in the form of fresh juice. [27]

ALA enhances the activities of vitamin C, E and Co-enzyme Q and it raises the level of Glutathione and thereby prevent the cell injury caused by reactive free radicals.

Lycophene

- Is an anti-oxidant from plants
- A red carotenoid source pigment
- Extracted from tomato.
- Considered to reduce risk of atherosclerosis, Myocardial Infarction and other cancers.
- Tetraterpenes – carotenoid.
- C_{40} yellow or orange red carotenoid pigment.
- Around 500 such compounds have been identified.
- They are formed by tail to tail union of 2 molecules of C_{20} geranylgeranyl diphosphate to give an acyclic intermediate with a cis – configuration of central double bond.
- By changing the above configuration to desaturating isophrenoid chain Lycopene.

Interferon - Gamma

It is Anti- Fibrotic cytokine.

It will alter the collagen synthesis

It is used intralesionally

Placental extract

Placental extract is having growth factors and Anti inflammatory agents. Anti inflammatory and anti-platelet activities are seen .

There are two important reasons for using placental grafts in OSMF

1. *hormonal effect,*
2. *mehanical effect.*

Hormonal: *Biogenic stimulant effect is because the placenta is a homograft, immunologically competent and rich in steroids, proteins, chorionic gonadotrophins, estrogens and progesterone.*

The grafts are easily mouldable and undergo total absorption only after prolonged periods, thus mechanically preventing fibrosis.

It is reported in studies,

Hyaluronidase acts upon the collagen and reduce the density of the intercellular cement substances, and it breaks down hyaluronic acid and prevents collagen formation.

As stated by Rajendran R et al.[39]High protein diet,Iodinated salt,Vitamins and Minerals are very essential in the management of OSMF.

XI.OTHER PREMALIGNANT AND MALIGNANT LESIONS OF ORAL CAVITY[40]

PREMALIGNANT LESIONS OF ORAL CAVITY

Following are the premalignant condition of Epithelial lining of oral mucosa which may turn to become squamous cell carcinoma.

1. Dysplasia.
2. Leukoplakia
3. Proliferative verrucous leukoplakia
4. Erythroplakia
5. Nicotine Stomatitis
6. Actinic Cheilitis
7. Smokeless tobacco keratosis
8. Lichen planus and
9. Melanosis and mucosal hyper pigmentation

1. Dysplasia

There will be alteration in Cell as dark staining nuclei and Nuclear, Cytoplasmic ratio altered

Loss of Mitotic activity,

Cellular maturation from basal cells to flattened superficial cells.

Carcinoma insitu Full or almost full thickness architectural abnormalities in viable cellular layers along with cytological atypia.

2. Leukoplakia

White patch or plaque that cannot be characterized clinically or pathologically as any other disease and is not associated with any physical or chemical causative agent except the use of tobacco.

- middle age and older population
- after 40 years

floor of month is the Most Common site in smokers.

WHO defined leukoplakia as a clinical white patch that cannot be characterized clinically or pathologically as any other disease. It is a clinical definition and does not take pathology into consideration.

Other white lesions of oral mucosa, 1. lichen planus,

2.discoid lupus erythematosus and

3.white spongy nevus .

Aetiology: smoking

tobacco chewing,

alcohol

Ill-fitting dentures and

cheek bites.

It may also be associated with submucous fibrosis, hyperplastic candidiasis or Plummer-Vinson syndrome.

Sites involved. Buccal mucosa

oral commissures

floor of mouth,

tongue,

gingivobuccal sulcus and

mucosal surface of lip.

Buccal mucosa is the most common site in India.

Age and Sex. Mostly, it is seen in the fourth decade, males are affected two to three times more often.

Clinical types

a) *Homogenous* - It is less often associated with malignancy.

b) *Nodular* variety -presents as white patches or nodules on erythematous base;

c) *Erosive (erythroleukoplakia)* it has erosions and fissures.and higher incidence of malignant transformation.

Histology. 25% of leukoplakias are having epithelial dysplasia from mild to severe. In High grade of dysplasia malignant change more.

Malignant potential. The chances of leukoplakia becoming malignant are about 5%. It varies according to the site and type of leukoplakia and the duration.

Management

1. Spontaneous healing when causative agent is removed.
2. Biopsy is taken to rule out malignancy.
3. surgical excision
4. ablation with laser or cryotherapy can be done.

3. Proliferative Verrucus leukoplakia (PVL)

It is a variant of leukoplakia with white mucosal plaques that develop nodular, papillary or verruciform surface projections.

Cause -unknown.

Tobacco usage not proved to be of cause.

4. Erythroplakia

Similar to leukoplakia, which is a white patch, erythroplakia is a red patch or plaque on the mucosal surface. Red colour is due to decreased keratinisation and as a result the red vascular connective tissue of the submucosa shines through.

There is no sex predilection.

common sites : lower alveolar mucosa,

gingivobuccal sulcus and the

floor of the mouth.

Most of lesions of erythroplakia show severe dysplasia, carcinoma in situ or a frank invasive carcinoma when first seen.

Malignant potential is 17 times higher than in leukoplakia.

Three types of lesions – 1.homogenous

2.speckled or granular, and

3.erythroplakia interspersed with areas of leukoplakia. Treatment is excision biopsy and follow up.

5. Nicotine Stomatitis

Nicotine form of keratosis occurring in palate

Commonly Seen in pipe or cigar smokers

It appears umbilicated papules with red central depression.

It is Common in Posterior region of hard palate and soft palates.

6. Actinic Chelitis

- Solar cheilosis
- Accelerated tissue degeneration of vermillion partion of lower lip due to prolonged exposure to sun.

7. Smokeless tobacco keratosis

- It is also called as snuff pouch or snuff dippers lesion.

Clinical feature is chronic white or gray translucent mucosal macula localized in areas of direct contact with smokeless tobacco.

Most common sites are mucobuccal fold of mandible or maxilla in either incisor or molar region.

Inter twinning their strands or streaks of white kurtosis of bilateral buccal mucosa.

8.Lichen planus :mainly involving buccal mucosa

9.Melanosis and mucosal hyperpigmentation Benign pigmented lesions of oral mucosa may transform into malignant melanomas; however, the incidence of this change is not known. About one-fourth of mucosal melanomas may resemble benign lesions and hence biopsy may become mandatory.

MALIGNANT LESIONS OF ORAL CAVITY

Carcinoma Oral Cavity

Aetiology

Compared to western countries, India has high incidence of oral cancers. Age adjusted incidence rate in India is 44.8 and 23.7 in males and females, respectively. Compared to 11.2 per 1,00,000 in USA. Several aetiological factors are responsible. (6-S aetiology, i.e. smoking, spirits, sharp jagged tooth, sepsis, syndrome of plummer – vinsion and syphilitic glossitis).

a) *Smoking*. Incidence of oral cancer is six times more in smokers than in non-smokers. In certain parts of India, there is an unusual habit of reverse smoking where burning end of the 'churat' (rolled tobacco leaf) is put in the mouth. This gives high incidence of cancer of the hard palate.

b) *Tobacco chewing.* Powdered tobacco, mixed with lime, is placed in some part of the vestibule of the mouth. Carcinoma develops at the site of the quid. Chewing "Pan" and keeping the quid in the vestibule is largely responsible for oral cancer in our country.

c) *Alcohol.* Cancer of upper aero-digestive tract occurs six times more in heavy drinkers as compared to non drinkers.

d) *Dietary deficiencies.* Their role in genesis of cancer has not been definitely established. Riboflavin deficiency may be responsible for cancer in alcoholics. Paterson-Brown-Kelly syndrome also called Plummer Vinson syndrome (iron deficiency anaemia) is responsible for cancer of the oral cavity and hypopharynx.

e) *Dental sepsis, jagged sharp teeth* and *ill fitting dentures,* all cause chronic irritation and may lead to development of cancer.

Sites of cancer in the lip and oral cavity are (AJCC 2002)

1. Mucosal lip (from junction of skin – vermilion border to line of contact of upper and lower lip)
2. Buccal mucosa (includes mucosa of cheek and inner surface of lips up to line of contact of opposing lip)
3. Anterior two thirds of tongue (Oral tongue)

4. Hard palate
5. Lower alveolar ridge
6. Upper alveolar ridge
7. Floor of mouth
8. Retromolar trigone.

Clinical presentation and treatment of cancer of the oral cavity at different sites are described below.

1. Carcinoma Lip

Mostly, it is squamous cell carcinoma, often seen in males in the age group of 40-70. Lower lip is more often involved. Site of predilection is between the midline and commissure of the lip. Lesion is of exophytic or ulcerative type. Lymph node metastases develop late. Submental and submandibular nodes are the first to be involved; other deep cervical nodes may also get involved later.

Treatment is surgical excision with adequate safety margin of healthy tissue and plastic repair of the defect. Lymph node metastases require block dissection.

Radiotherapy also gives good results in early cases. Buccal mucosa covers a large area. It extends from the meeting point of lips in front to the

pterygomandibular raphe behind and from upper gingivobuccal sulcus to the lower one.

Carcino of buccal mucosa is very common. Its incidence is next only to tongue cancer. Equally seen in both sexes.

Site of origin: Most common site is the angle of mouth or the line of occlusion of upper and lower teeth. It may also arise from the buccal sulcus where 'pan' or tobacco quid is kept. As the whole of buccal mucosa is "condemned", carcinoma may be multicentric.

Gross appearance. Lesion may be exophytic or ulceroinfiltrative; the latter may infiltrate deeply. Exophytic type may be associated with erythroleukoplakia. Buccal mucosa is also the most common site for verrucous carcinoma which is a white papillary growth with considerable keratinisation.

Local spread. From its site of origin, the lesion may spread *deeply* involving submucosa → muscle → subcutaneous fat → skin. Involvement of buccinators muscle or anterior masseter causes trismus.

Tumour may spread radially from its site of origin and involve angle of the mouth and lip anteriorly, retromolar trigone and medial pterygoid

posteriorly, upper gingi vobuccal sulcus and maxilla superiorly, lower gingivobuccal sulcus and alveolar ridge and gums inferiorly.

3. Carcinoma Oral Tongu

Carcinoma involving anterior two thirds of tongue is commonly seen in men in the age group of 50-70 years. It may also occur in younger age group and in females. It may also develop on a pre-existing leukoplakia, long standing dental ulcer or syphilitic glossitis. Vast majority are squamous cell type.

Site. Most common site is middle of the lateral border or the ventral aspect of the tongue. Uncommonly, the tip or the dorsum may be involved.

Spread. Locally, it may infiltrate deeply into the lingual musculature causing ankyloglossia or may spread to the floor of mouth, alveolus and mandible. Lymph node metastases go to the submandibular and upper jugular nodes (from the lateral border of tongue) and to the submental and jugulo omohyoid group (from the tip). Bilateral or contralateral nodal involvement can also occur.

Clinically, cancer of the oral tongue presents as:

i) An exophytic lesion like a papilloma.

ii) A non-healing ulcer with rolled edges, grayish white shaggy base and induration.

iii) A submucous nodule with induration of the surrounding tissue.

Stage III or IV tumours require combined treatment with surgery and post operative radiotherapy. It gives better results than either modality alone. Block dissection neck is always done.

Depending on the size and extent of the primary lesion of the tongue, surgery may consist of hemiglossectomy including a portion of the floor of mouth, segment or hemimandibulectomy and block dissection of neck nodes the so-called “commando operation”.

4. Carcinoma Hard Palate

It is either squamous cell or variety; the latter being more common. Glandular variety arises from minor salivary glands of the palate and may be adenoid cystic, mucoepidermoid or adenocarcinoma. It is common in our country especially in people who have the habit of reverse smoking, i.e., keeping the burning end of bidi or cigar in the mouth. Both men and women are affected.

Cancer starts as a superficial ulcer with rolled out edges and gives no symptoms except painless irregularity on the palate felt by the tongue. It may spread to the gingival, lip, soft palate or invade the bone of hard palate, floor of the nasal cavity or the antrum. Lymphatic metastases may spread to the submandibular and upper jugular nodes. Cancer palate should be differentiated from cancer of maxillary antrum or nose which has spread to the palate.

5. Carcinoma of Alveolar Ridges

It is also called gingival carcinoma; it is mostly seen in men. Usual site of involvement is lower jaw behind the first molar. Tumour may spread to the check, floor of mouth, retromolar trigone or the hard palate. Gingival cancer may invade the underlying bone and then spread rapidly along the neurovascular bundle. Nodal metastases go to submandibular and upper jugular nodes.

6. Cancer Floor of Mouth

Squamous cell carcinoma is the most common. It affects males more than females in ratio of 4:1. Typically, lesions start anteriorly near the opening of submandibular duct which may get obstructed, leading to enlargement of submandibular gland.

Usually, the lesion is ulcerative or infiltrative type and spreads locally into the adjoining areas such as ventral aspect of the tongue, lingual gingival, mandibular periosteum or deeply into the floor of mouth and submental space. Lymphatic metastases go to submandibular nodes. Lesions of the floor of mouth remain asymptomatic for a long time or cause soreness or irregularity in the floor of the mouth. A swelling in the submandibular region may be either due to obstructive enlargement of submandibular salivary gland or lymph node metastases and this may require differentiation.

7. Carcinoma Retromolar Trigone

Involvement of retromolar trigone may be primary, or secondary to extension of growths from the gingiva, floor of mouth, buccal mucosa or the palatine arch.

Treatment depends on the extent of lesion. Wide surgical excision often combined with block dissection is required.

5. MATERIALS AND METHODS

Study Design: Randomised Controlled Trail.

Settings and Subjects: This study includes the patients having newly diagnosed and histopathologically confirmed Oral submucous fibrosis (OSMF) in Thanjavur Medical College Hospital ,Thanjavur during the period from January 2011 to July 2012.

Sample Size: All the eligible patients according to the inclusion and exclusion criteria within the study period will form the study population. It is expected to be around 60.

Inclusion criteria:

1. Both males and females.

2. Age 13 and above.

Exclusion criteria:

1. Patients with other pre cancerous and cancerous lesions of oral cavity.

2. Severe Trismus, Inter incisor distance less than 1cm.

Outcome Measures:

1. Sociodemographic details of Oral submucous fibrosis (OSMF) patients.

2. Habits of the patients which are the risk factors of Oral submucous fibrosis OSMF have been collected by the history.

3. Pre-treatment signs and symptoms are assessed.

4. Pre-treatment, post-treatment signs and symptoms are compared.

Data Collection: data collected using pre-tested structured questionnaire.

Data Entry and Analysis:

The Data collected will be entered in a Excel Spreadsheet and will be analyzed using statistical software SPSS Version 16. Mean, SD will be calculated; 't' test, Chi ^{2} Test and other appropriate statistical tests will be applied.

Randomised Control Trail was designed to conduct in our Hospital after the protocol was approved by the ethical committee.

The study consisted totally 60 patients clinically diagnosed and biopsy proved as OSMF. Patients with other oral mucosal disorders, systemic disorders or those who are not willing to participate in this study were excluded. The patients were randomly divided into two groups of 30each, the case –group 1 and control –group 2.

Each patient was interviewed in detail, with the particular reference to the types ,frequency and duration of abusive habits related to the oral cavity

.the symptoms and signs were recorded using a 0-4 points visual analogue score (VAS).

Patients were counselled

- To stop chewing,
- To avoid unbranded irritant tooth powders,
- To stop using sharp sticks
- To use branded tooth paste with their own fingers so that the mucosa may not get injured by brush

Normal mouth opening (interincisor distance)

Males -5.25 cm

Females -4.75 cm

Clinically this can be measured by Putting patients own fingers vertically. Normal individual can admit his four fingers vertically.

GROUP I: (ALPHA LIPOIC ACID GROUP) patients in this group were treated with ALPHA LIPOIC ACID orally once daily for 12 weeks in adjunct with steroid injection of HYDROCORTISONE SODIUM 100mg and HYALURONIDASE 1500 IU were given intralesionally as multiple puncture weekly once for 12 weeks.

GROUPII: (NON-ALPHA LIPOIC ACID GROUP) patients in this group were treated with steroid injection of HYDROCORTISONE SODIUM 100mgand HYALURONIDASE 1500 IU were given intralesionally as multiple puncture weekly once for 12 weeks .

All the patients were followed up for 6 months to one year after completion of treatment in both groups. There were no dropouts in this study and they were all well aware of the fact that they were treated for oral pre-cancerous condition.

Each patient from these two groups was evaluated every week for clinical improvement .Signs and symptoms were compared before the treatment and after treatment.

This study includes the patients having newly diagnosed and histopathologically confirmed Oral submucous fibrosis .Most of the patients although initially agreed for a post-treatment biopsy, refused biopsy after completion of treatment.

Mangal singh and H.S. Niranjan in their study *quoted that "Two groups were compared by using interincisor distance and Histopathological improvement. This kind of study has not been done so far."*[42]

Once diagnosis was made, the treatment was started immediately without delay.

6. RESULTS AND OBSERVATION

6.1 General Characteristics of the Study Population

TABLE 6.1.1

AGE DISTRIBUTION

AGE GROUP	GROUP		TOTAL
	1 (ALA GROUP)	2 (NON-ALA GROUP)	
Up to 20 Yrs	1 (3.3%)	4 (13.3%)	5 (8.3%)
21- 30 Yrs	6 (20.0%)	6 (20.0%)	12 (20.0%)
31- 40 Yrs	9 (30.0%)	9 (30.0%)	18 (30.0%)
41- 50 Yrs	11 (36.7%)	7 (23.3%)	18 (30.0%)
51- 60 Yrs	3 (10.0%)	4 (14.3%)	7 (11.7%)
Total	30 (100%)	30 (100%)	60 (100%)

Chi^2 – 2.832 df – 4 p-Value – 0.586

Minimum age of the study group – 14 yrs; Maximum age of the study group – 60 yrs; Mean age of group I – 39 yrs; Mean age of group II -37 yrs; and Over all Mean - 38.30 yrs.

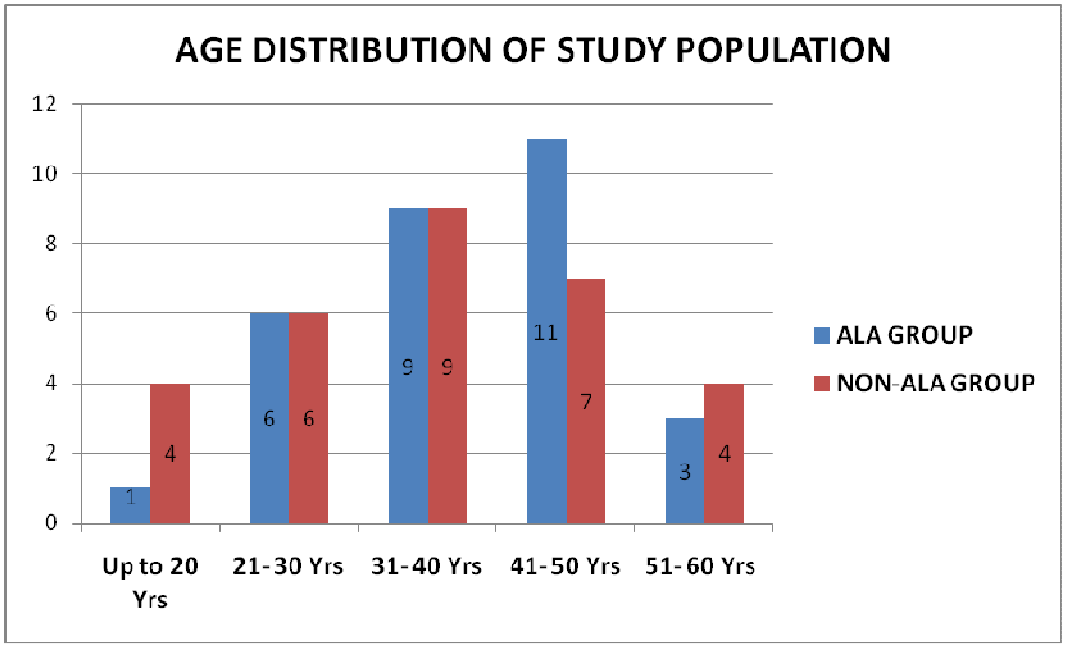


FIGURE 6.1.1

MULTIPLE BAR CHART SHOWING AGE DISTRIBUTION OF THE STUDY POPULATION

TABLE 6.1.2

SEX DISTRIBUTION

SEX	GROUP		TOTAL
	1	2	
MALE	12 (40.0%)	17 (56.7%)	29 (48.3%)
FEMALE	18 (60.0%)	13 (43.3%)	31 (51.7%)
Total	30 (100%)	30 (100%)	60 (100%)

Chi^2 – 1.669 df – 1 p-Value – 0.196

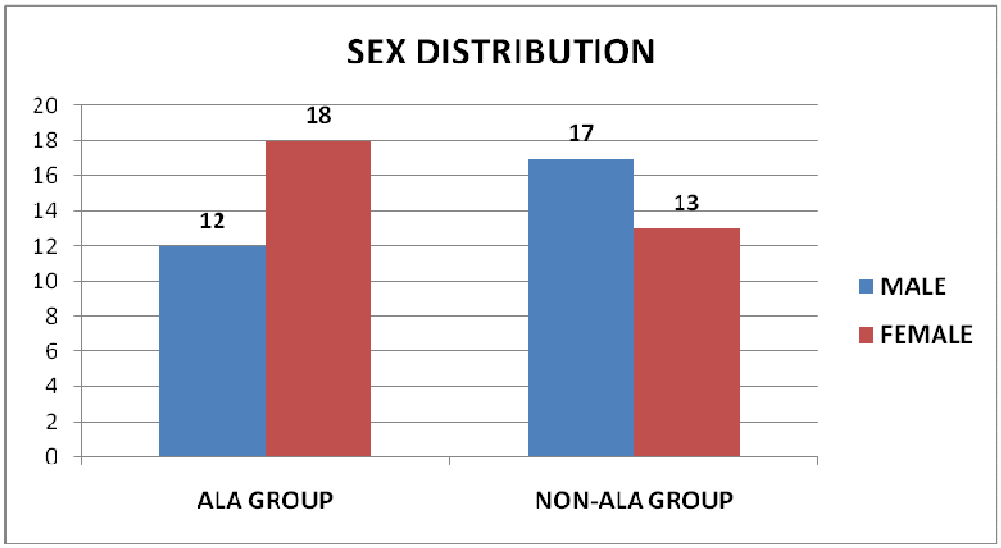


Fig. 6.1.2

MULTIPLE BAR CHART SHOWING SEX DISTRIBUTION

6.1.3 EDUCATION

EDUCATION GROUP	GROUP		TOTAL
	1	2	
NIL	5 (16.7%)	7 (23.3%)	12 (20.0%)
PRIMARY	14 (46.7%)	7 (23.3%)	21 (35.0%)
MIDDLE	2 (6.7%)	2 (6.7%)	4 (6.7%)
HIGH	3 (10.0%)	11 (36.7%)	14 (23.3%)
HIGHER SECONDARY	3 (10.0%)	1 (3.3%)	4 (6.7%)
DEGREE	3 (10.0%)	2 (6.7%)	5 (8.3%)
Total	30 (100%)	30 (100%)	60 (100%)

Chi^2 – 8.438 df – 5 p-Value – 0.134

6.2 CHARACTERISTICS RELATED TO OSMF

TABLE 6.2.1

PERSONAL HABITS

PERSONAL HABITS	GROUP		TOTAL	Chi^2 - Value	p-Value
	1	2			
TOBACCO	9 (30.0%)	7 (23.3%)	16 (26.7%)	0.341	0.559
BETELNUT	3 (10.0%)	1 (3.3%)	4 (6.7%)	1.071	0.301
BL,BN,LIME	17 (56.7%)	11 (36.7%)	28 (46.7%)	2.411	0.121
PAN MASALA	4 (13.3%)	7 (23.3%)	11 (18.3%)	1.002	0.317
FABRICATED	5 (20.0%)	11 (36.7%)	17 (28.3%)	2.052	0.152
ALCOHOL	2 (6.7%)	1 (3.3%)	3 (5.0%)	0.351	0.554
SMOKING	6 (20.0%)	5 (16.7%)	11 (18.3%)	0.111	0.739

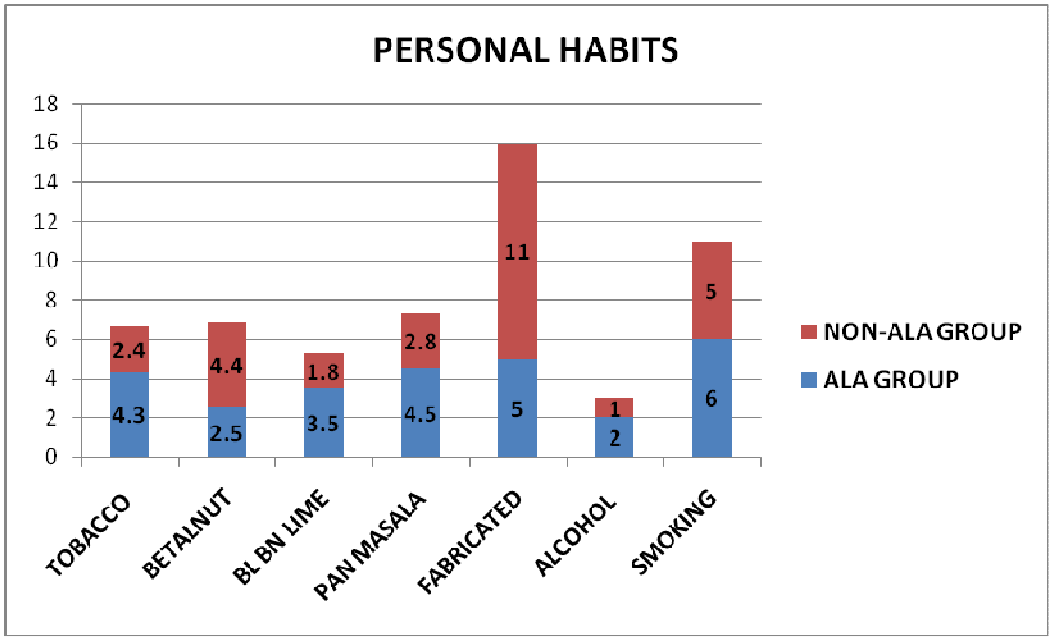


FIG. 6.2.1

COMPONENT BAR CHART SHOWING THE PERSONAL HABITS OF THE STUDY POPULATION

TABLE 6.2.2

SYMPTOMS RELATED TO OSMF

SYMPTOMS	GROUP		TOTAL	Chi^2 Value	p-Value
	1	2			
BURNING	28 (93.3%)	29 (96.7%)	57 (95.0%)	0.351	0.554
DRYNESS	27 (90.0%)	29 (96.7%)	56 (93.3%)	0.278	0.302
TRISMUS	30 (100%)	30 (100%)	60 (100%)	-	-
BLANCHING	30 (100%)	30 (100%)	60 (100%)	-	-
FIBROUS BAND	28 (93.3%)	30 (100%)	58 (96.7%)	0.523	0.472
ANKYLOGLOSSIA	26 (86.7%)	26 (86.7%)	52 (86.7%)	0.000	1.000
STOMATITIS	24 (80.0%)	25 (83.3%)	49 (81.7%)	0.111	0.739
SOFT PALATE	25 (83.3%)	24 (80.0%)	49 (81.7%)	0.137	0.718

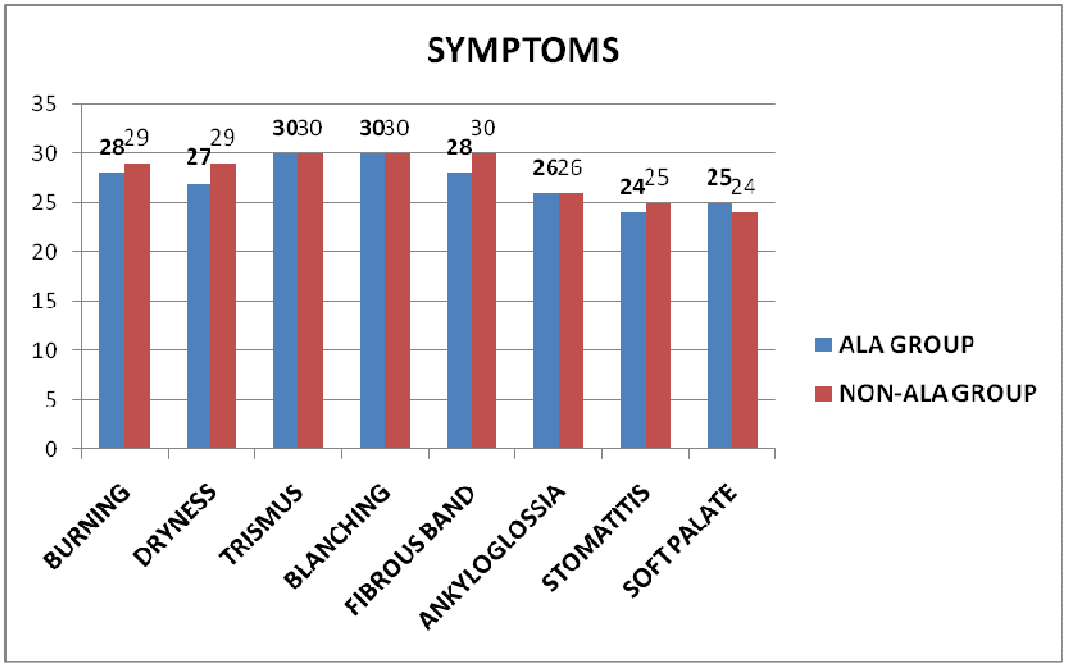


FIGURE 6.2.2.

MULTIPLE BAR CHART SHOWING SYMPTOMS OF OSMF IN BOTH GROUPS

TABLE 6.2.3

ORAL HYGIENE

ORAL HYGIENE	GROUP		TOTAL
	1	2	
POOR	15 (50.0%)	17 (56.7%)	32 (53.3%)
FAIR	13 (43.3%)	11 (36.7%)	24 (40.0%)
GOOD	2 (6.7%)	2 (6.7%)	4 (6.7%)
Total	30 (100%)	30 (100%)	60 (100%)

Chi^2 – 0.292 df – 2 p-Value – 0.864

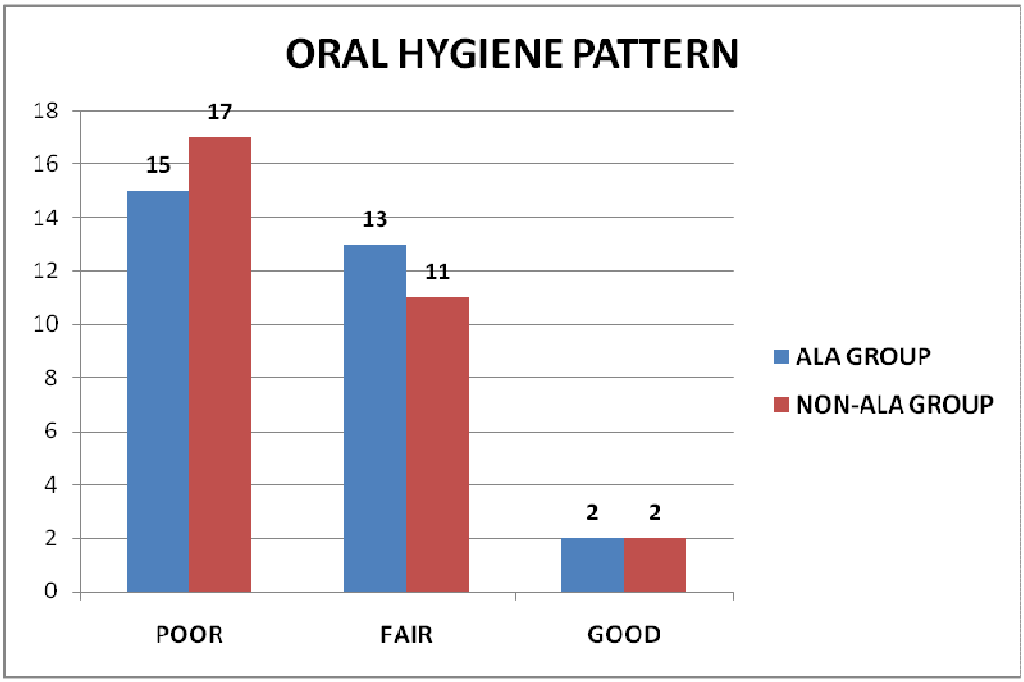


FIGURE 6.2.3

MULTIPLE BAR CHART SHOWING ORAL HYGIENE PATTERN IN BOTH GROUPS

TABLE 6.2.4

MATERIAL FOR BRUSHING TEETH

MATERIAL FOR BRUSHING TEETH	GROUP		TOTAL
	1	2	
SHARP STICK	7 (23.3%)	4 (13.3%)	11 (18.3%)
TOOTH PASTE	11 (36.7%)	11 (36.7%)	22 (36.7%)
UNBRANDED	12 (40.0%)	15 (50.0%)	27 (45.0%)
Total	30 (100%)	30 (100%)	60 (100%)

Chi^2 – 1.152 df – 2 p-Value – 0.562

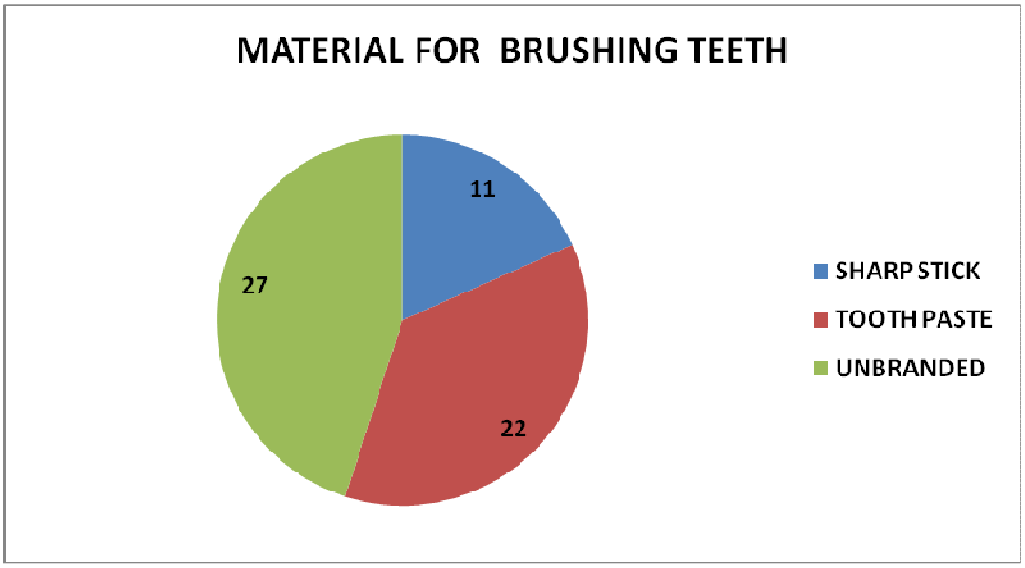


FIGURE 6.2.4

PIE CHART SHOWING THE MATERIALS

USED FOR BRUSHING TEETH

TABLE 6.3

DESCRIPTIVE STATISTICS

	GROUP 1	GROUP 2	OVERALL	F - Value	p - Value
AGE IN YEARS	39.23 ± 10.54	37.37 ± 12.66	38.30 ± 11.59	0.385	0.537
DISTANCE in Kms	23.33 ± 13.88	19.73 ± 15.49	21.48 ± 14.17	0.913	0.343
INCOME in Rs	4208.33 ± 4036.52	4616 ± 2935.17	4412.50 ± 3505.09	0.201	0.656
HB in mg%	8.98 ± 1.48	8.38 ± 1.44	8.68 ± 1.48	2.573	0.114
TOTAL COUNT	6768 ± 2230	6615 ± 2827	6692 ± 2525	0.055	0.816

TABLE 6.4

MEAN PRE- TREATMENT SCORE

	GROUP 1	GROUP 2	OVERALL	F - Value	p - Value
PRE BURNING	3.00 ± 1.15	3.20 ± 0.66	3.10 ± 0.93	0.685	0.411
PRE DRYNESS	2.97 ± 1.13	3.20 ± 0.66	3.08 ± 0.93	0.952	0.333
PRE TRISMUS	3.30 ± 0.79	3.40 ± 0.56	3.35 ± 0.68	0.316	0.576
PRE BLANCHING	3.47 ± 0.63	3.47 ± 0.62	3.43 ± 0.62	0.171	0.681
PRE FIBROUS	3.33 ± 0.88	3.20 ± 0.81	3.27 ± 0.84	0.371	0.544
PRE ANKYLOGLOSSIA	3.00 ± 1.11	2.53 ± 1.52	2.77 ± 1.35	1.831	0.181
PRE STOMATITIS	2.73 ± 1.36	2.33 ± 1.54	2.53 ± 1.46	1.136	0.291
PRE SOFT PALATE	2.70 ± 1.34	2.20 ± 1.49	2.45 ± 1.43	1.857	0.178
PRE TOTAL	24.50 ± 6.04	23.47 ± 5.92	23.98 ± 5.98	0.447	0.506

TABLE 6.5 PAIRED T-TEST FOR GROUP 1

		Mean	Std. Deviation	t - Value	Significance
Pair 1	PRE BURNING	3.00	1.145	9.607	0.000*
	POST BURNING	1.57	.935		
Pair 2	PRE DRYNESS	2.97	1.129	9.424	0.000*
	POST DRYNESS	1.57	.935		
Pair 3	PRE TRISMUS	3.30	.794	9.633	0.000*
	POST TRISMUS	1.97	.809		
Pair 4	PRE BLANCHING	3.47	.629	11.366	0.000*
	POST BLANCHING	2.07	.868		
Pair 5	PRE FIBROUS	3.33	.884	10.420	0.000*
	POST FIBROUS	1.97	.999		
Pair 6	PRE ANKYLOGLOSSIA	3.00	1.114	6.901	0.000*
	POST ANKYLOGLOSSIA	1.87	1.137		
Pair 7	PRE STOMATITIS	2.73	1.363	6.998	0.000*
	POST STOMATIS	1.70	1.179		
Pair 8	PRE SOFT PALATE	2.70	1.343	6.595	0.000*
	POST SOFT PALATE	1.70	1.179		
Pair 9	PRETOTAL	24.50	6.044	12.103	0.000*
	POSTTOTAL	14.43	6.585		

* Statistically Significant (p -Value < 0.001)

There is statistically significant difference in all the individual symptoms and overall symptoms between pre treatment and post treatment of alpha lipoic acid group (group I), Statistically Significant (p -Value < 0.001).

TABLE 6.6

PAIRED T-Test FOR GROUP 2

		Mean	Std. Deviation	t - Value	Significance
Pair 1	PRE BURNING	3.20	.664	10.933	0.001*
	POST BURNING	1.90	.712		
Pair 2	PRE DRYNESS	3.20	.664	10.933	0.001*
	POST DRYNESS	1.90	.712		
Pair 3	PRE TRISMUS	3.40	.563	11.948	0.002*
	POST TRISMUS	2.10	.662		
Pair 4	PRE BLANCHING	3.40	.621	11.894	0.000*
	POST BLANCHING	2.13	.681		
Pair 5	PRE FIBROUS	3.20	.805	10.865	0.000*
	POST FIBROUS	2.07	.740		
Pair 6	PRE ANKYLOGLOSSIA	2.53	1.525	4.805	0.000*
	POST ANKYLOGLOSSIA	1.70	1.088		
Pair 7	PRE STOMATITIS	2.33	1.539	4.955	0.000*
	POST STOMATIS	1.43	1.104		
Pair 8	PRE SOFT PALATE	2.20	1.495	4.817	0.000*
	POST SOFT PALATE	1.53	1.042		
Pair 9	PRETOTAL	23.47	5.923	13.310	0.000*
	POSTTOTAL	14.87	5.532		

* Statistically Significant (p -Value < 0.05)

TABLE 6.7

COMPARISON OF DIFFERENCE IN SIGNS AND SYMPTOMS SCORE BETWEEN TWO GROUPS

	GROUP 1	GROUP 2	OVERALL	F - Value	p - Value
DIF BURNING	1.43 ± 0.82	1.07 ± 0.52	1.25 ± 0.71	4.295	0.043*
DIF DRYNESS	1.40 ± 0.81	0.90 ± 0.31	1.15 ± 0.66	9.932	0.003*
DIF TRISMUS	1.33 ± 0.76	1.00 ± 0.37	1.17 ± 0.62	4.677	0.035*
DIF BLANCHING	1.40 ± 0.66	1.00 ± 0.37	1.20 ± 0.56	8.093	0.006*
DIF FIBROUS	1.37 ± 0.72	0.90 ± 0.61	1.13 ± 0.70	7.382	0.009*
DIF ANKYLOGLOSSIA	1.23 ± 0.86	0.57 ± 0.57	0.90 ± 0.79	12.581	0.001*
DIF STOMATITIS	1.03 ± 0.81	0.57 ± 0.63	0.80 ± 0.76	6.246	0.015*
DIF SOFT PALATE	1.00 ± 0.83	0.47 ± 0.51	0.73 ± 0.73	9.010	0.004*
DIF TOTAL	10.13 ± 4.58	7.53 ± 3.91	8.83 ± 4.42	5.596	0.021*

* Statistically Significant (p -Value < 0.05)

There is statistically significant difference in all the individual symptoms and overall symptoms between alpha lipoic acid group (group I) and Non-alpha lipoic acid group. Statistically Significant (p -Value < 0.05)

7. DISCUSSION

60 cases in this study are in age group of 13 to 60, with majority of them fell in 40-50 years of life.

Minimum age of the study group is 14 ,Maximum age of the study group – 60,Mean age of group I – 39,Mean age of group II -37 ,And the Overall mean age -38.30

Out of 60 cases 29were males and 31 were females, Males and females are almost equal in numbers.

Only 6.7% studied up to higher secondary school, 35% of them studied up to primary school, 5% are studied in college, and 20% of them are uneducated.

There is no significant difference in education pattern among two groups.

p-Value >0.05

There is no significant difference in education pattern among two groups.

p-Value >0.05 table 6.1.3

Personal habits are closely related with this disease. Following habits were observed 16 patients have taken tobacco, 4 patients have taken betel nut, and 28 patients have taken betel leaf, betel nut and lime. 11 patients have taken

pan masala, 17 patients have taken fabricated betel nut, 3 patients have taken alcohol and 11 were smokers .As it is seen in the table 6.2.1 these personal habits are almost equally distributed in both groups. p-Value >0.05.

This study includes the patients having newly diagnosed and histopathologically confirmed Oral submucous fibrosis .Most of the patients although initially agreed for a post-treatment biopsy, refused biopsy after completion of treatment.

86.7% of people in this study population were having Angular Stomatitis is the state of nutritional deficiency. It may synergize the disease condition in contributing to epithelial atrophy.

Almost all the patients in the study population were chewing betel nut in various forms. Majority of them chewed betel nut with betel leaf and lime.

All the patients in this study were having trismus and blanching of oral mucosa, and 96.7% of them were having fibrous band and 93.3% having dryness of mouth.

Out of 60 patients only 4 patients were having good oral hygiene; it is 6.4% in the study group, 53.3% of them having poor oral hygiene and 40% of them having fair oral hygiene.

In this study nearly 50% of the patients were using unbranded powders for brushing their teeth. This will irritate the mucosa and may aggravate the disease process, and 18.3% of patients were using sharp sticks,

Mean value of Age of the patient, Distance from health care, Income, Haemoglobin% and Total White Blood Cell count were studied in this study.

There is no difference in distribution of the mean value of these above factors between two groups

Symptoms and signs were observed in this study group,95%of the total study group had burning sensation, 93.3% -dryness of mouth, and 100% ,that is all 60 patients presented with trismus and blanched mucosa, 96.7% of patients had fibrous band, 86.7% had ankyloglossia, 81.7% had angular stomatitis and 81.7% had soft palate involvement. There is no significant difference in prevalence of symptoms and signs were observed in both groups as p-Value >0.05.

With available studies these symptoms and signs were seen in more than 80% of study groups.

Oral hygiene is the index of the severity of OSMF; only 4 patients were having good oral hygiene, it is 6.4% in the study group, 53.3% of them having poor oral hygiene and 40% of them having fair oral hygiene. Oral hygiene is also equally distributed in both groups. There is no significant difference between two groups. P-Value >0.05.

In this study nearly half of the patients were using unbranded powders for brushing their teeth. This will irritate the mucosa and may aggravate the disease process. and 18.3% of patient were using sharp sticks ,these sharp ends

of the stick will produce more and more damage to mucosa as evident in table 6.2.4 and there is no significant difference were observed between two groups p-Value >0.05.

In this study Age of the patient, Distance from health care, Income, Hemoglobin% and total WBC count were also taken into consideration. There is no difference in distribution of the mean value of these above factors between two groups.

We can assume that all the factors mentioned above are not playing their role while interpreting the treatment outcome of these two groups.

In table 6.4 mean pre-treatment signs and symptoms score between group 1 and group 2 were compared. And found that both these group are very much comparable before treatment.

Pre-treatment and post-treatment signs and symptoms score of group 1 compared with paired t-test. And it revealed that, all the individual signs and symptoms and overall signs and symptoms showed highly significant difference between pre-treatment and post-treatment score. It is shown in table 6.5 p-Value <0.001.

Based on the result given in the table 6.5 Alpha lipoic acid is highly effective when used with injection hydrocortisone sodium and hyaluronidase.

While comparing Pre-treatment and post-treatment signs and symptoms score of group 2 compared with paired t-test, highly significant difference

noted in Blanching, Fibrous Band, Ankyloglossia and Angular Stomatitis. (p-Value <0.001)

And in Burning sensation Dryness of mouth and Trismus were significant only (p-value <0.01) .it shows that group 2(Non ALAgroup) is also effective but not to the level where ALA group as it is evident by p-value. This observation is confirmed by the table 6.6

Difference in the signs and symptoms of pre-treatment and post treatment between the two groups were compared, there is statistically significant difference in all the signs and symptoms of pre-treatment and post treatment score between alpha lipoic acid group (group I) and Non-alpha lipoic acid group. Statistically Significant (p -Value < 0.05) according to the table 6.7

The following should be implemented by the Government the Ministry of social welfare and other organizations and also by medical professionals to prevent and control the progression of disease. The operation should be started at the root.

1. Where products are manufactured. Generally they are small scale industries, run by people of a locality. They should be trained for their earning in some other job.
2. Where these plants are cultivated. Other than a small number of people for whom betel, tobacco plant cultivation is the major occupation, large group of people do this as an accessory work in their fields where they grow trees, palm casuarinas etc.

3. The channel through which they are transported and sold.
4. Sale near residential and educational places should be banned.
5. Prohibition in cinemas, mass education regarding adverse effects. In town areas, passing information, communication and educating people is easier than in villages where social workers should go and talk to a gathering, in schools and in places where people gather during function and festival.
6. So as for smoking, strict rules should be implemented for pan masala, tobacco chewing and spitting in public places like hospitals, cinema theatres and from running buses on road side.

8. CONCLUSION

To conclude, both educated and uneducated people are getting affected knowingly or unknowingly. Even if some people realize, they could not come out from this habit due to addiction.

Areca nut and tobacco are the precipitating factors. Therefore preventive measures must be taken

These products are neither bearing proper labels nor the health warnings.

Pre-treatment and post-treatment signs and symptoms score of ALA-Group compared with paired t-test. And it revealed that, all the individual signs and symptoms and overall sign and symptoms were highly significant.

In younger individuals, popularity of commercially available products are on the rise rather than traditional betel quid, leading to adoption of this habit at a younger age.

Even though the educated people in the study populations are habitual chewers, there is a need for proper health education in our community.

According to the table 6.3 nearly 20% of study populations are not educated and only 8.3% are having degree and only 35% of them studied up to primary school.

There is definite improvement in the symptom especially burning sensation of mouth and mouth opening that is interincisor distance measured

with a caliber in all patients who were included in this study. Mean Pre-treatment trismus score in group I is -3.3 has became reduced after treatment as 1.97,whereas Mean Pre-treatment trismus score in group II is 3.4 has become reduced after treatment as 2.1 It reveals the efficacy of the therapy to the patient.

And at the end of the study a significant difference between the two groups were observed .Even though there is clinical improvement in both these groups, ALA group is proved to be better than Non ALA group. A statistically significant difference has been observed.

In our hospital set up,

- we colleagues advice and also make patients who come with tobacco stained tooth, in earlier stage of the disease, to stop this habit which is only 50% fruitful with our maximum care.
- Last, but not the least, there should be individual awareness and motivation to restrain from this habit.

The antioxidant Alpha lipoic acid, has a definitive protective role as demonstrated in this study, and it can certainly be recommended for clinical use as routine.

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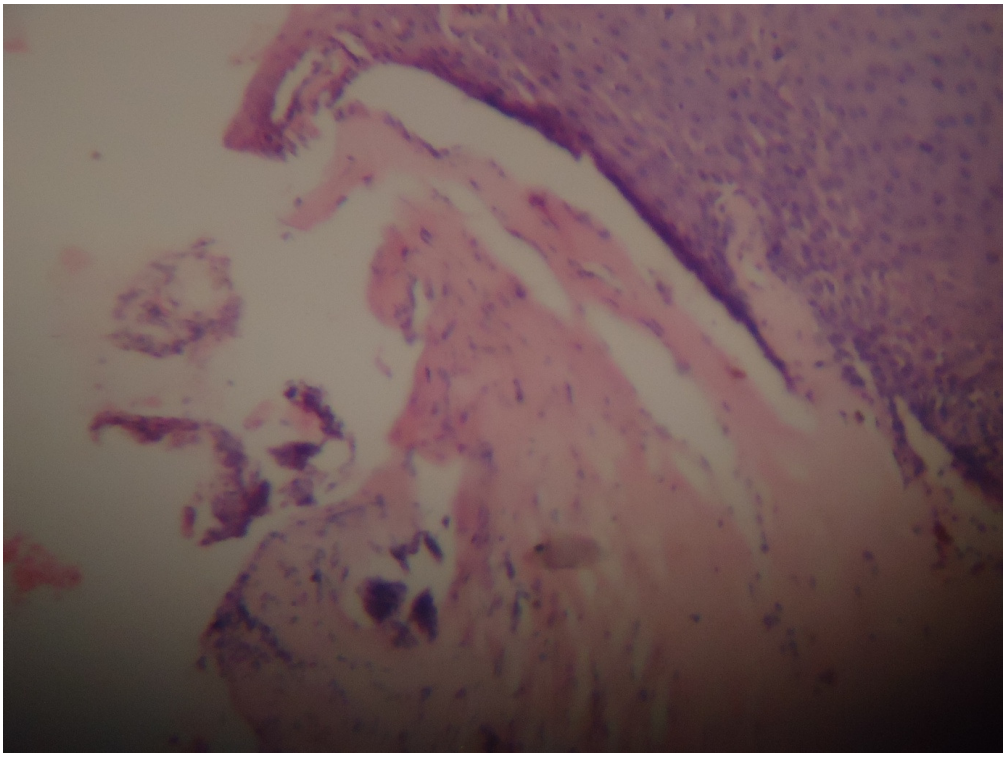

Figure 4.9

GRADE 3-section shows minimal sub epithelial fibroblast proliferation and Collagen is moderately hyalinised, and blood vessels are constricted

.

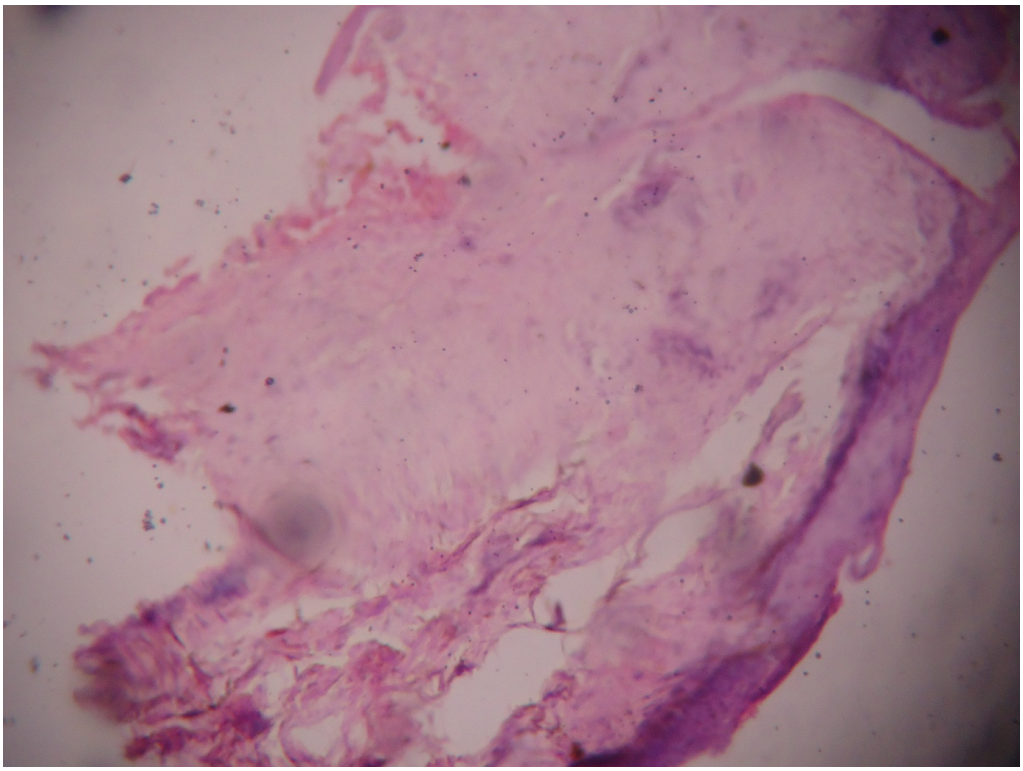

Figure 4.8

GRADE 2- section shows hyperplastic squamous epithelium, sub-epithelium shows fibroblastic proliferation with collagen deposition.

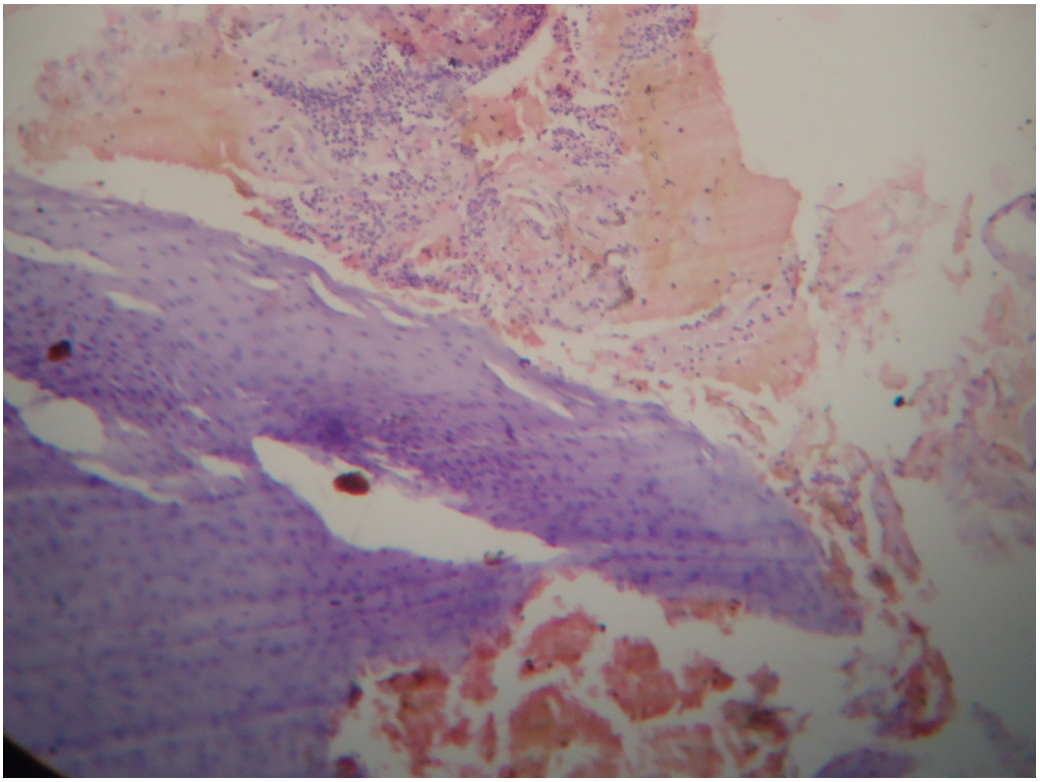

Figure 4.7

GRADE 1-section shows hyperplastic squamous epithelium, sub-epithelium shows acute inflammatory cells, minimal fibroblastic proliferation and congested blood vessels.

Figure 4.10

MARBLE LIKE APPEARANCE OF ORAL MUCOSA IN A PATIENT WITH OSMF

Figure 4.11

TRISMUS IN A 19 YRS OLD CHRONIC CHEWER

Figure 4.12

THE ABOVE SAID PATIENT WITH MARBLE COLOURED BUCCAL MUCOSA INVOLVING SOFT PALATE , TONSILLAR PILLARS

Figure 4.2

CONICAL / ROUNDED BROWNISH UNPROCESSED FRESH FORM OF BETEL NUT

Figure 4.4

SHARP EDGES CAUSE MORE DAMAGE TO MUCOSA,
FORCE NEEDED TO CRUSH THE HARD NUTS

Figure 4.3

NATURALLY AVAILABLE FORM OF BETEL LEAF, BETEL NUT AND TOBACCO, IMPROPERLY LABELLED SWEETENED PACKED FORM OF BETEL NUT.

Figure 4.5

PACKED TOBACCO AND GUTKHA-IN ATTRACTIVE FORM AVAILABLE IN THE MARKET

Figure 4.13

CALIBER USED TO MEASURE INTERINCISOR DISTANCE

IN A CASE OF OSMF

Figure 4.14

WOODEN PAMBARAM USED FOR MOUTH OPENING EXERCISE

Figure 4.6

DEPARTMENTAL SHOPS SELLING PROCESSED PACKED TOBACCO

ALONG WITH OTHER NUTRITIONAL SUBSTANCES

NORMAL ORAL MUCOSA

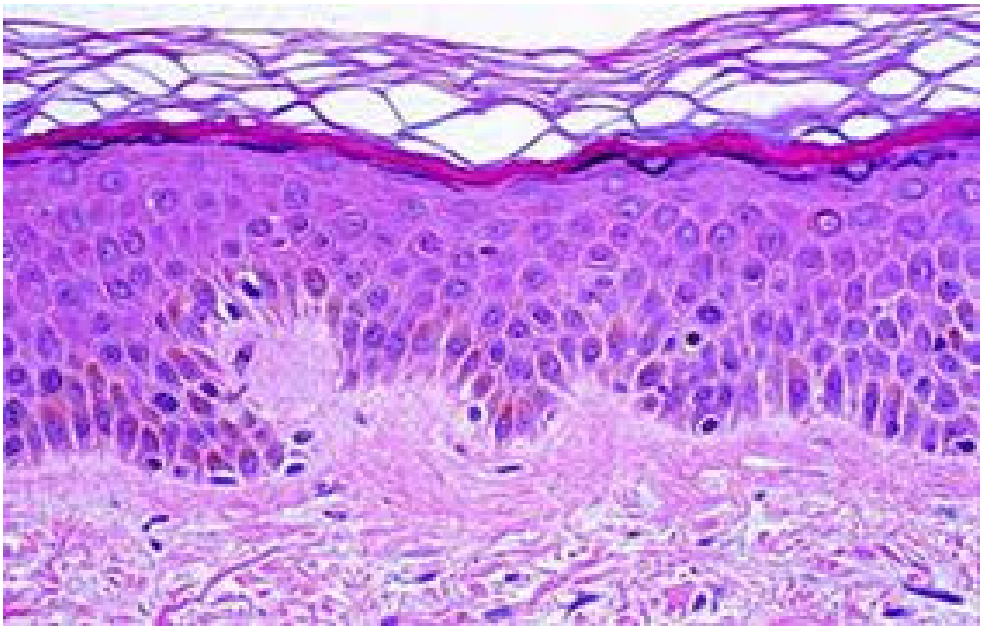

Figure 4.1(A)

NORMAL ORAL MUCOSA-STRATIFIED SQUAMOUS EPITHELIUM SUPPORTED BY A LAMINA PROPRIA, UNDER THE MUCOSA THERE IS TOUGH SUBMUCOSAL LAYER WITH SALIVARY GLANDS.

Figure 4.15

INTRALESIONAL INJECTION HYDROCORTISONE AND HYALURONIDASE BEING INJECTED TO A PATIENT WITH OSMF

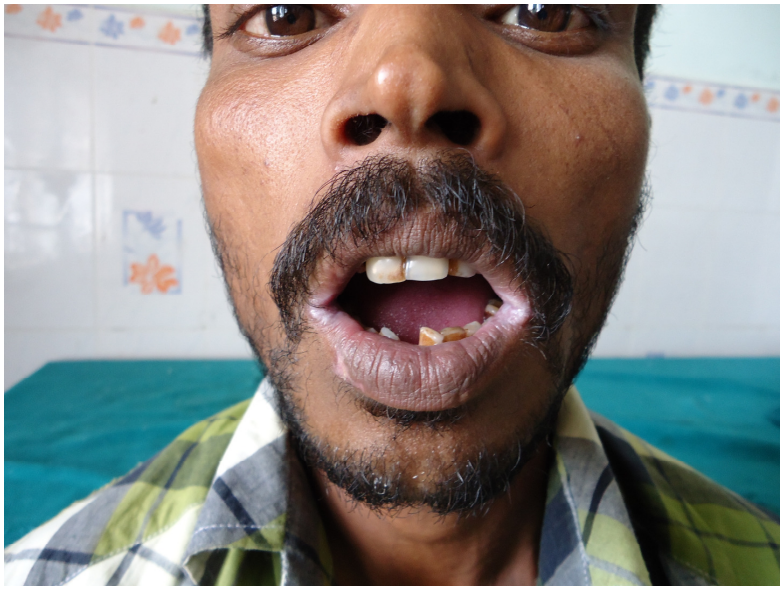

Figure 4.17
PRE-TREATMENT PICTIRE OF A PATIENT WITH OSMF SHOWING TRISMUS AND ANGULAR STOMATITIS

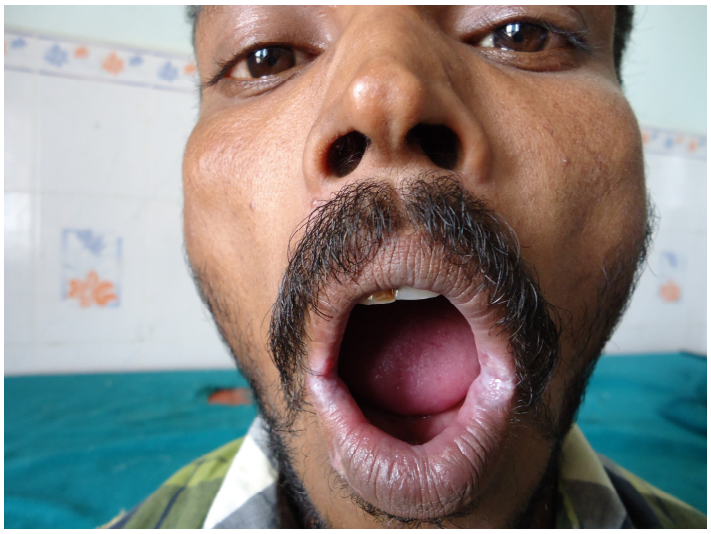

Figure 4.18
POST TREATMENT PICTURE OF THE PATIENT SHOWING IMPROVEMENT IN TRISMUS

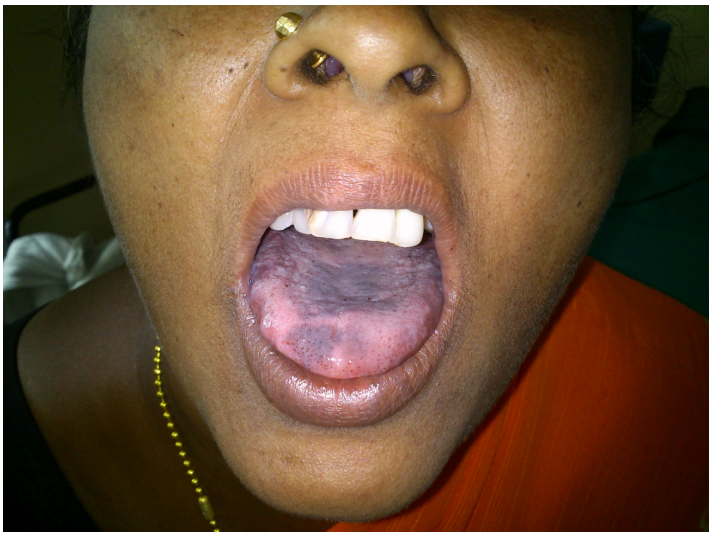

Figure 4.19

PRE-TREATMENT PICTIRE OF A PATIENT WITH OSMF SHOWING TRISMUS AND HYPER PIGMENTATION

Figure 4.20

POST TREATMENT PICTURE OF THE PATIENT SHOWING IMPROVEMENT IN TRISMUS

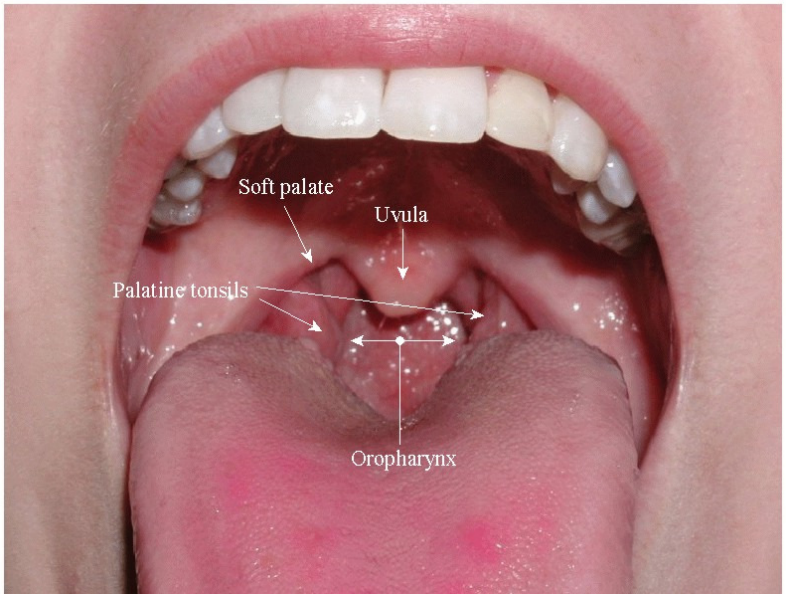

Figure 4.1(B)

ORAL CAVITY AND OROPHARYNX

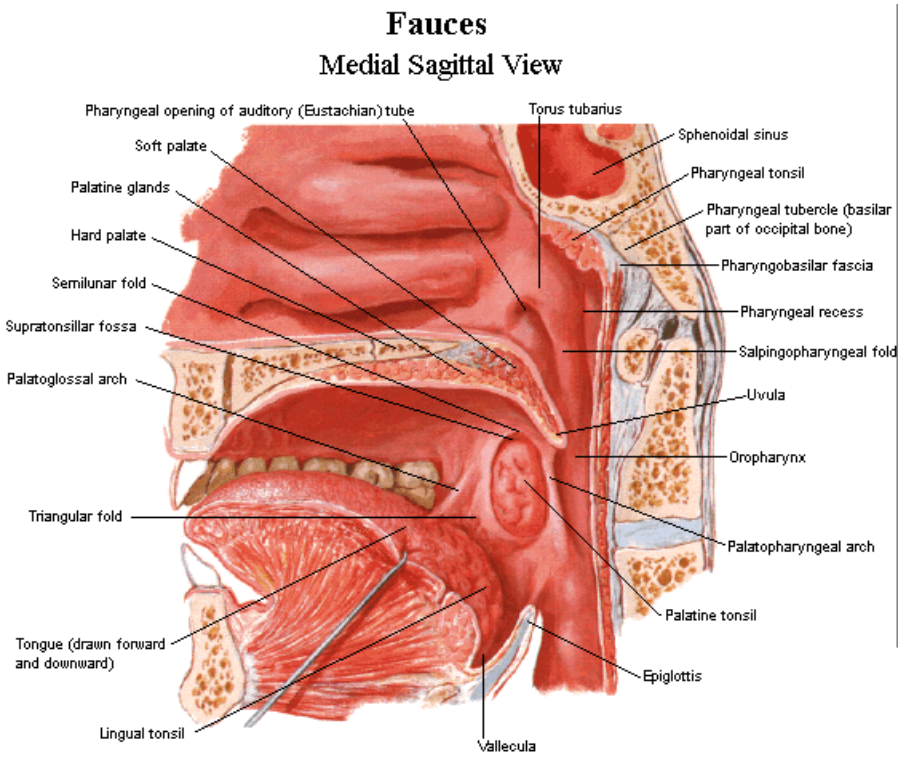

Figure 4.1(C)

MEDIAL SAGITTAL VIEW OF OROPHARYNX

Figure 4.16
INJECTION SITES

OSMF - MASTER CHART

S.NO	GROUP	NAME	AGE	SEX	DISTANCE	EDUCATION	EDUCATION GROUP	OCCUPATION	INCOME	TOBACCO	BETELNUT	BLBNLIME	PANMASALA	FABRICATED B.N	ALCOHOL	SMOKING	BURNING	DRYNESS	TRISMUS	BLANCHING	FIBROUSBAND	ANKYLOGLOSSIA	STOMATITIS	SOFT PALATE
1	1	VALAVAMBAL	60	FEMALE	25	NIL	NIL	COOLIE	2,000			3,3					Y	Y	Y	Y	N	Y	N	N
2	1	VINAYAKI	48	FEMALE	25	NIL	NIL	COOLIE	2,000		24,6						Y	Y	Y	Y	Y	Y	Y	Y
3	1	KADALMANI	35	FEMALE	30	NIL	NIL	COOLIE	2,000			15,5					Y	Y	Y	Y	Y	Y	Y	Y
4	1	GANDHIMATHI	41	FEMALE	10	5	PRIMARY	H.W	0			3,5					Y	Y	Y	Y	Y	Y	Y	Y
5	1	SHAKILA	45	FEMALE	50	5	PRIMARY	H.W	0	2,5		2,5					Y	Y	Y	Y	Y	Y	Y	Y
6	1	NAGAMMAL	30	FEMALE	15	4	PRIMARY	COOLIE	3,000					3,5			N	N	Y	Y	Y	Y	Y	Y
7	1	ANJAMMAL	45	FEMALE	35	2	PRIMARY	VENDOR	10,000					10			Y	Y	Y	Y	Y	Y	Y	Y
8	1	INDRANI	45	FEMALE	25	NIL	NIL	COOLIE	3,000	25,5		25,5					Y	Y	Y	Y	Y	Y	Y	Y
9	1	PAPPA	55	FEMALE	17	NIL	NIL	COOLIE	3,500			30,5					Y	Y	Y	Y	Y	Y	Y	Y
10	1	SIVABAKIYAM	50	FEMALE	32	2	PRIMARY	COOLIE	4,500	8,5		8,5					Y	Y	Y	Y	Y	Y	Y	Y
11	1	PARVATHY	45	FEMALE	16	5	PRIMARY	COOLIE	3,000	20,5		20,5					Y	Y	Y	Y	Y	Y	Y	Y
12	1	UTHIRAPATHY	34	MALE	19	8	MIDDLE	TAILOR	8,000				3,10		12	12,5	Y	Y	Y	Y	Y	Y	Y	Y
13	1	SELVI	39	FEMALE	20	5	PRIMARY	COOLIE	3,000	20,5		20,5					Y	Y	Y	Y	Y	Y	Y	Y
14	1	KAMALAHASAN	15	MALE	65	8	MIDDLE	H.SERVER	2,750				3,10				Y	Y	Y	Y	Y	Y	Y	Y
15	1	NEELAVATHY	40	FEMALE	18	3	PRIMARY	COOLIE	3,500					10,10			Y	Y	Y	Y	Y	Y	Y	Y
16	1	BASHARAN	50	MALE	10	PUC	DEGREE	SALES MAN	20,000	20,5	20,5						Y	Y	Y	Y	Y	Y	Y	Y
17	1	PREM	29	MALE	56	12	HIGER SECONDARY	BUS CON	5,000				2,10				Y	Y	Y	Y	Y	Y	N	N
18	1	YESURAJ	22	MALE	7	12	HIGER SECONDARY	COOLIE	5,000					2,10			Y	N	Y	Y	Y	Y	Y	Y
19	1	SUNDARAMBAL	27	FEMALE	22	5	PRIMARY	COOLIE	5,000					2,10			Y	Y	Y	Y	Y	Y	Y	Y
20	1	RAJENDRAN	45	MALE	23	5	PRIMARY	COOLIE	7,000	20,5	20,5						Y	Y	Y	Y	Y	Y	Y	Y
21	1	DURAIMANIKAM	34	MALE	25	5	PRIMARY	COOLIE	4,500			10,5			10	10,5	Y	Y	Y	Y	Y	N	N	N
22	1	PRABU	28	MALE	30	BBA	DEGREE	STUDENT	0				1				Y	Y	Y	Y	Y	N	N	N
23	1	MAHALAKSMI	37	FEMALE	15	10	HIGH	HW	0			15,10					Y	Y	Y	Y	Y	Y	Y	Y
24	1	VASANTHA	45	FEMALE	29	3	PRIMARY	COOLIE	4,500			25,8					Y	Y	Y	Y	Y	Y	Y	Y
25	1	ARUN KARTHI	22	MALE	22	DECE	DEGREE	STUDENT	0					1		1	Y	Y	Y	Y	N	N	N	N
26	1	DHARMARAJ	55	MALE	12	10	HIGH	COOLIE	5,000			,30,10				20,24	N	N	Y	Y	Y	Y	Y	Y
27	1	RANI	39	FEMALE	5	3	PRIMARY	HW	0	10,10		10,10					Y	Y	Y	Y	Y	Y	Y	Y
28	1	SUNDARARAJAN	35	MALE	15	10	HIGH	COOLIE	10,000			10,5				10,10	Y	Y	Y	Y	Y	Y	Y	Y
29	1	RAMESH	37	MALE	15	12	HIGER SECONDARY	WATCH MAN	5,000	17,5		17,5				10,5	Y	Y	Y	Y	Y	Y	Y	Y
30	1	MEENA	45	FEMALE	9	5	PRIMARY	COOLIE	5,000			20,7					Y	Y	Y	Y	Y	N	N	N

OSMF - MASTER CHART

S.NO	NAME	ORAL HYGIENGE	BRUSHING MATERIAL	HB	TC	DCP	DCL	DCM	DCE	PREBURNING	PREDRYNESS	PRETRISMUS	PREBLANCHING	PREFIBROUS	PREANKYLOGLOSSI	PRESTOMATITIS	PRESOFTPALATE	POSTBURNING	POSTDRYNESS	POSTTRISMUS	POSTBLANCHING	POSTFIBROUS	POSTANKYLOGLOSSI	POSTSTOMATIS	POSTSOFTPALATE
1	VALAVAMBAL	POOR	UNBRANDED	11	6,700	69	27			3	3	1	3	0	3	0	0	1	1	1	1	0	1	0	0
2	VINAYAKI	POOR	UNBRANDED	9	6,200	67	29		4	4	4	3	3	3	3	3	3	1	1	1	1	1	1	1	1
3	KADALMANI	POOR	UNBRANDED	9	7,400	66	31		3	3	3	3	3	3	3	3	3	1	1	2	2	2	2	2	2
4	GANDHIMATHI	POOR	SHARP STICK	8	6,500	63	33		4	3	3	4	4	4	4	4	4	2	2	3	3	3	3	3	3
5	SHAKILA	FAIR	TOOTH PASTE	10	4,000	56	41	3		4	4	4	4	4	3	3	3	2	2	3	3	3	3	2	2
6	NAGAMMAL	FAIR	SHARP STICK	10	7,500	69	28		3	0	0	3	3	3	2	2	2	0	0	2	2	2	2	2	2
7	ANJAMMAL	FAIR	TOOTH PASTE	9	3,400	57	40		3	4	4	4	4	4	3	4	4	1	1	2	2	2	2	2	2
8	INDRANI	POOR	SHARP STICK	10	6,400	56	40		4	3	3	3	3	3	3	3	3	2	2	2	2	2	2	2	2
9	PAPPA	POOR	UNBRANDED	7	9,600	70	26		4	3	3	3	4	4	4	4	4	1	1	3	4	4	4	4	4
10	SIVABAKIYAM	FAIR	TOOTH PASTE	10	7,000	68	30		2	4	4	4	4	4	3	4	4	3	3	3	3	3	3	3	3
11	PARVATHY	POOR	UNBRANDED	13	6,200	58	38		4	3	3	2	3	3	3	2	2	1	1	1	1	1	1	1	1
12	UTHIRAPATHY	FAIR	TOOTH PASTE	12	6,200	58	38	4		4	3	4	4	4	4	4	4	2	2	2	2	2	2	2	2
13	SELVI	POOR	UNBRANDED	10	6,700	61	36	3		4	4	4	4	4	4	4	4	3	3	3	3	3	3	3	3
14	KAMALAHASAN	FAIR	TOOTH PASTE	8	8,000	65	22		3	3	3	3	3	3	3	3	3	1	1	1	1	1	1	1	1
15	NEELAVATHY	POOR	SHARP STICK	10	6,800	62	25		3	3	3	3	4	4	4	4	4	1	1	2	2	2	3	3	3
16	BASHARAN	GOOD	TOOTH PASTE	9	8,480	69	28		3	2	2	2	2	2	1	1	1	1	1	1	1	1	1	1	1
17	PREM	FAIR	TOOTH PASTE	8	14,500	85	10		5	3	3	4	4	4	4	0	0	2	2	2	2	1	1	0	0
18	YESURAJ	FAIR	TOOTH PASTE	8	9,500	70	26		4	0	0	3	3	4	4	4	3	0	0	2	2	3	3	3	3
19	SUNDARAMBAL	FAIR	UNBRANDED	8	5,000	56	40		4	3	3	4	4	4	4	4	4	2	2	2	2	2	1	1	1
20	RAJENDRAN	POOR	UNBRANDED	9	5,800	57	40		3	4	4	4	4	3	3	3	3	3	3	2	2	2	1	1	1
21	DURAIMANIKAM	FAIR	SHARP STICK	8	5,000	58	48		4	3	3	4	4	3	1	1	1	1	1	1	3	2	0	0	0
22	PRABU	FAIR	TOOTH PASTE	10	6,280	65	32		3	3	3	2	2	2	0	0	0	2	2	2	2	0	0	0	0
23	MAHALAKSMI	POOR	UNBRANDED	8	5,400	60	37		3	4	4	4	4	4	4	4	4	2	2	3	3	3	3	3	3
24	VASANTHA	POOR	UNBRANDED	9	4,200	58	39		3	3	3	3	3	3	4	4	4	1	1	1	1	2	2	2	2
25	ARUN KARTHI	GOOD	TOOTH PASTE	8	5,000	58	39		3	3	3	3	3	3	2	2	2	1	1	1	1	1	1	1	1
26	DHARMARAJ	FAIR	SHARP STICK	9	5,000	62	35		3	0	0	3	3	3	3	3	3	0	0	1	1	1	1	1	1
27	RANI	POOR	UNBRANDED	6	5,000	61	36		3	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4
28	SUNDARARAJAN	FAIR	TOOTH PASTE	7	6,200	59	37		4	3	3	3	3	3	4	2	2	2	2	2	2	2	3	1	1
29	RAMESH	POOR	SHARP STICK	10	10,600	64	32		4	3	3	4	4	4	2	2	2	2	2	2	2	2	2	2	2
30	MEENA	POOR	UNBRANDED	11	8,500	57	40		3	4	4	4	4	4	1	1	1	2	2	2	2	2	0	0	0

OSMF - MASTER CHART

S.NO	GROUP	NAME	AGE	SEX	DISTANCE	EDUCATION	EDUCATION GROUP	OCCUPATION	INCOME	TOBACCO	BETELNUT	BLBNLIME	PANMASALA	FABRICATED B.N	ALCOHOL	SMOKING	BURNING	DRYNESS	TRISMUS	BLANCHING	FIBROUSBAND	ANKYLOGLOSSIA	STOMATITIS	SOFT PALATE
31	2	MARIMUTHU	30	MALE	20	5	PRIMARY	COOLIE	5,000	5,5		5,5					Y	Y	Y	Y	Y	Y	Y	Y
32	2	RAMAN	60	MALE	3	5	PRIMARY	COOLIE	5,000	35,5		35,5					Y	Y	Y	Y	Y	Y	Y	Y
33	2	CHANDRASEKAR	20	MALE	25	9	HIGH	COOLIE	5,000					2,5			Y	Y	Y	Y	Y	Y	Y	Y
34	2	SANTHANAMERY	40	FEMALE	5	3	PRIMARY	SALES WOMAN	5,000					5,10			Y	Y	Y	Y	Y	Y	Y	Y
35	2	MUTHUPANDI	14	MALE	4	8	MIDDLE	STUDENT	0					2,5			Y	Y	Y	Y	Y	Y	Y	Y
36	2	SUNDARARAJAN	55	MALE	5	NIL	NIL	COOLIE	5,000	20,5		20,5					Y	Y	Y	Y	Y	Y	N	N
37	2	BAKIARAJ	33	MALE	10	10	HIGH	BUS CON	7,500		3,5						Y	Y	Y	Y	Y	N	N	N
38	2	VASUKI	44	FEMALE	25	NIL	NIL	COOLIE	4,000			30,10					Y	Y	Y	Y	Y	N	N	N
39	2	MARIMUTHU	30	MALE	25	MA	DEGREE	LECTURER	12,000					5,5		5,10	Y	Y	Y	Y	Y	N	N	N
40	2	GANGHIMATHI	50	FEMALE	65	NIL	NIL	HW	0			20,5					Y	Y	Y	Y	Y	Y	Y	N
41	2	KARTHIK	29	MALE	20	10	HIGH	CORPENDER	10,000				4,4			4,4	Y	Y	Y	Y	Y	Y	Y	Y
42	2	SUGUMAR	31	MALE	6	4	PRIMARY	COOLIE	6,000				12,5				Y	Y	Y	Y	Y	Y	Y	Y
43	2	KASIAMMAL	50	FEMALE	20	NIL	NIL	COOLIE	3,500	25,10							Y	Y	Y	Y	Y	Y	Y	Y
44	2	MARUTHAMBAL	50	FEMALE	5	NIL	NIL	COOLIE	3,000	10,10							Y	Y	Y	Y	Y	Y	Y	Y
45	2	LAKSMI	47	FEMALE	20	10	HIGH	COOLIE	3,500					5,10			Y	Y	Y	Y	Y	Y	Y	Y
46	2	VIJAYA	40	FEMALE	12	5	PRIMARY	COOLIE	4,000			10,5					Y	Y	Y	Y	Y	Y	Y	Y
47	2	PRABU	29	MALE	20	10	HIGH	VENDOR	5,000				3,6			7,6	Y	Y	Y	Y	Y	Y	Y	Y
48	2	SARAN	15	MALE	5	9	HIGH	STUDENT	0					2,10			Y	Y	Y	Y	Y	Y	Y	Y
49	2	MAHAMUNY	37	MALE	25	10	HIGH	H.SERVER	5,000			10,6					Y	Y	Y	Y	Y	Y	Y	Y
50	2	KALYANASUNDRAM	24	MALE	28	BSC	DEGREE	TEACHER	7,500				5,20				Y	Y	Y	Y	Y	N	N	Y
51	2	VIJAYALAKSMI	38	FEMALE	30	12	HIGER SECONDARY	VENDOR	6,000			11,5					Y	Y	Y	Y	Y	Y	Y	Y
52	2	CHANDRA	40	FEMALE	25	5	PRIMARY	COOLIE	3,000					5,10			Y	Y	Y	Y	Y	Y	Y	Y
53	2	BALASARSWATHY	35	FEMALE	23	9	HIGH	HW	0					5,10			Y	Y	Y	Y	Y	Y	Y	Y
54	2	SELVAM	33	MALE	9	10	HIGH	MECHANIC	5,000				3,10	3,10			Y	Y	Y	Y	Y	Y	Y	Y
55	2	PRAKASH	15	MALE	25	9	HIGH	STUDENT	0				2,5	2,5			Y	Y	Y	Y	Y	Y	Y	Y
56	2	MUTHUKANNU	50	FEMALE	25	NIL	NIL	COOLIE	4,500	25,5		25,5					Y	Y	Y	Y	Y	Y	Y	Y
57	2	RUKMANI	55	FEMALE	25	NIL	NIL	COOLIE	4,000			15,5					Y	Y	Y	Y	Y	Y	Y	Y
58	2	KULANTHIRAJ	52	MALE	10	6	MIDDLE	CORPENDER	10,000				5,5			5,5	Y	Y	Y	Y	Y	Y	Y	Y
59	2	THANGARAJ	46	MALE	60	10	HIGH	COOLIE	5,000	25,10		25,10			20	30,10	N	Y	Y	Y	Y	Y	Y	Y
60	2	ANJALAI	29	FEMALE	12	5	PRIMARY	COOLIE	5,000					5,5			Y	Y	Y	Y	Y	Y	Y	Y

OSMF - MASTER CHART

S.NO	NAME	ORAL HYGIENGE	BRUSHING MATERIAL	HB	TC	DCP	DCL	DCM	DCE	PREBURNING	PREDRYNESS	PRETRISMUS	PREBLANCHING	PREFIBROUS	PREANKYLOGLOSSIA	PRESTOMATITIS	PRESOFTPALATE	POSTBURNING	POSTDRYNESS	POSTTRISMUS	POSTBLANCHING	POSTFIBROUS	POSTANKYLOGLOSSL	POSTSTOMATIS	POSTSOFTPALATE
31	MARIMUTHU	FAIR	TOOTH PASTE	7	7,500	68	28		4	4	4	3	3	3	4	2	1	2	2	2	2	2	2	1	1
32	RAMAN	POOR	UNBRANDED	8	6,600	50	45		5	3	3	3	3	3	3	3	3	2	2	2	2	2	2	2	2
33	CHANDRASEKAR	FAIR	TOOTH PASTE	8	3,300	69	27		4	3	3	3	3	3	3	3	3	2	2	2	2	2	2	2	2
34	SANTHANAMERY	FAIR	UNBRANDED	10	3,000	68	28		4	4	4	4	4	4	4	4	4	2	2	2	2	2	2	2	2
35	MUTHUPANDI	FAIR	TOOTH PASTE	7	6,500	58	40		2	3	3	4	4	4	4	4	4	2	2	3	3	3	3	3	3
36	SUNDARARAJAN	POOR	SHARP STICK	10	5,000	50	48		2	3	3	3	3	3	4	0	0	2	2	2	2	2	2	0	0
37	BAKIARAJ	FAIR	TOOTH PASTE	9	7,000	58	40		2	3	3	4	2	2	0	0	0	1	1	1	1	1	0	0	0
38	VASUKI	FAIR	UNBRANDED	6	10,800	70	28		2	3	3	4	3	3	0	0	0	2	2	2	2	2	0	0	0
39	MARIMUTHU	GOOD	TOOTH PASTE	7	5,000	51	45		4	3	3	4	4	4	0	0	0	3	3	3	3	3	0	0	0
40	GANGHIMATHI	POOR	UNBRANDED	8	5,600	51	44		5	3	3	3	4	4	4	4	1	2	2	2	3	3	3	3	1
41	KARTHIK	POOR	UNBRANDED	8	3,000	50	48		2	3	3	4	4	4	4	4	4	2	2	2	2	2	2	2	2
42	SUGUMAR	POOR	UNBRANDED	8	5,000	63	34		3	4	4	4	4	4	4	4	4	3	3	3	3	3	3	3	3
43	KASIAMMAL	POOR	SHARP STICK	10	11,600	42	57		1	4	4	4	4	4	4	4	4	2	2	3	3	3	3	3	3
44	MARUTHAMBAL	POOR	SHARP STICK	8	6,400	57	40		3	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4
45	LAKSMI	POOR	UNBRANDED	8	3,200	66	31		3	3	3	3	3	3	3	3	3	1	1	2	2	2	2	1	1
46	VIJAYA	POOR	UNBRANDED	11	13,500	50	48		2	3	3	3	3	3	1	1	3	1	1	1	1	1	1	1	1
47	PRABU	FAIR	TOOTH PASTE	9	3,500	52	45		3	3	3	3	3	1	1	1	1	1	1	2	2	1	1	1	1
48	SARAN	FAIR	TOOTH PASTE	8	3,800	69	28		3	3	3	3	3	3	2	1	1	1	1	2	2	2	1	1	1
49	MAHAMUNY	FAIR	TOOTH PASTE	8	5,600	66	32		2	3	3	4	4	4	4	4	4	1	1	2	2	2	3	1	2
50	KALYANASUNDRAM	GOOD	TOOTH PASTE	11	8,550	60	38		2	3	3	3	3	3	1	1	1	2	2	2	2	2	1	1	1
51	VIJAYALAKSMI	FAIR	TOOTH PASTE	9	6,500	58	40		2	3	3	3	3	4	0	0	1	2	2	2	2	2	1	0	1
52	CHANDRA	FAIR	SHARP STICK	6	5,800	65	30		5	3	3	3	3	2	4	4	4	2	2	2	2	2	3	3	3
53	BALASARSWATHY	POOR	UNBRANDED	7	6,400	57	40		3	4	4	4	4	4	4	4	4	3	3	3	3	3	0	0	3
54	SELVAM	POOR	TOOTH PASTE	10	6,600	71	24		5	3	3	3	3	3	2	2	2	2	2	2	2	2	1	1	1
55	PRAKASH	POOR	UNBRANDED	8	3,300	69	27		4	1	1	3	4	3	1	2	1	1	1	1	1	1	1	1	1
56	MUTHUKANNU	POOR	UNBRANDED	10	7,600	76	20		4	4	4	4	4	2	1	2	1	2	2	2	2	1	1	1	1
57	RUKMANI	POOR	UNBRANDED	6	12,000	58	40		2	4	4	3	4	3	4	4	3	2	2	2	2	2	3	2	2
58	KULANTHIRAJ	POOR	UNBRANDED	7	10,800	70	28		2	2	2	2	2	2	2	2	2	1	1	1	1	1	1	1	1
59	THANGARAJ	POOR	UNBRANDED	9	7,200	65	31		4	3	3	4	4	4	2	2	2	2	2	2	2	2	2	2	2
60	ANJALAI	POOR	UNBRANDED	10	7,800	68	28		4	4	4	3	3	3	2	1	1	2	2	2	2	2	1	1	1

Thanjavur Medical College

THANJAVUR, TAMILNADU, INDIA-613004

(Affiliated to the T.N Dr.MGR Medical University, Chennai)

ETHICAL COMMITTEE

CERTIFICATE

Name of the Candidate	:	Dr.A. AMIRTHAGANI
Course	:	M.S ENT
Period of Study	:	APRIL 2010- APRIL 2013
College	:	THANJAVUR MEDICAL COLLEGE
Dissertation Topic	:	EFFICACY OF ALPHA LIPOIC ACID IN ADJUNCT WITH INTRALESIONAL HYDROCORTISONE AND HYALURONIDASE IN THE MANAGEMENT OF ORAL SUBMUCOUS FIBROSIS.

The Ethical Committee, Thanjavur Medical College has decided to inform that your Dissertation Topic is accepted and you are permitted to proceed with the above study.

11.9.12

Thanjavur

Date :

Secretary

Ethical Committee

QUESTIONNAIRE FOR THE STUDY ON ORAL SUBMUCOUS FIBROSIS

NAME OF THE PATIENT :
AGE :
ADDRESS :

OP/IP NO :
SEX :

DISTANCE FROM THE MEDICAL FACILITY :
EDUCATION :
OCCUPATION :
MONTHLY INCOME :

HABITS	**DURATION**	**QUANTITY**	**FREQUENCY**
TOBACCO ALONE			
TOBACCO WITH BETEL NUT			
BETEL LEAF WITH BETAL NUT WITH LIME			
PAN MASALA			
FABRICATED BETEL NUT			
ALCOHOL			
SMOKING			
SPICY FOOD			

CLINICAL SYMPTOMS	YES	NO
BURNING SENSATION ON TAKING FOOD		
DRYNESS OF MOUTH		

CLINICAL SIGNS	YES	NO
TRISMUS		
BLANCHING		
FIBROUS BAND		
ANKYLOGLOSSIA		
STOMATITIS		
SOFT PALATE INVOLVEMENT		

ORAL HYGIENE: POOR FAIR GOOD

MATERIAL USED FOR BRUSHING: TOOTH PASTE/ SHARP STICKS/UNBRANDED POWDER

LAB INVESTIGATIONS

Hb % :

TC : , DC- P %, L %, M %, E %.

PRE TREATMENT - SYMPTOMS SCORE	0	1	2	3	4
BURNING SENSATION ON TAKING FOOD					
DRYNESS OF MOUTH					

PRE TREATMENT - SIGNS SCORE	0	1	2	3	4
TRISMUS					
BLANCHING					
FIBROUS BAND					
ANKYLOGLOSSIA					
STOMATITIS					
SOFT PALATE INVOLVEMENT					

POST TREATMENT - SYMPTOMS SCORE	0	1	2	3	4
BURNING SENSATION ON TAKING FOOD					
DRYNESS OF MOUTH					

POST TREATMENT - SIGNS SCORE	0	1	2	3	4
TRISMUS					
BLANCHING					
FIBROUS BAND					
ANKYLOGLOSSIA					
STOMATITIS					
SOFT PALATE INVOLVEMENT					

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Assignment title	Medical
Author	Amirthagani 22102221 M.S. ENT
E-mail	amirthaganijoseph@yahoo.com
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First 100 words of your submission

1. INTRODUCTION Oral sub mucous fibrosis is a precancerous lesion, predominant condition seen in South Asian ethnic group ,more in India .No age or sex predilection has been found. OSMF is primarily a cell mediated immune reaction to areca nut chewing and betel quid. It is prevalent in people belonging to poor socioeconomic status associated with nutritional deficiency. In 1953 S.G.Joshi termed this condition as Oral sub mucous fibrosis. And latter it was described by Pindborg and Sirsat in (1966). 1 In simple words Oral sub mucous fibrosis is a chronic progressive crippling fibrotic but preventable disease. Manifestations are dryness and burning sensation in the mouth, blanching of oral...